

Original Research Article

Neurocomputational Decoding of Feigned Psychosis: Frontoparietal Cognitive Control Activation and Support Vector Machine Classification for Objective Forensic Symptom Validity Assessment in Indonesia

Taufiq Indera Jayadi¹, Taryudi Suharyana^{2*}, Vita Amanda³, Brenda Jaleel⁴¹Department of Radiology, Phlox Institute, Palembang, Indonesia²Department of Neurology, CMHC Research Center, Palembang, Indonesia³Department of Psychiatry, CMHC Research Center, Palembang, Indonesia⁴Department of Neuroscience, San Fernando General Hospital, San Fernando, Trinidad and Tobago

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Corresponding author

Taryudi Suharyana

E-mail:

taryudi.suharyana@cattleyacenter.id

ORCID: 0009-0000-2367-0670

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ABSTRACT

Background. Detecting feigned psychiatric symptoms is a high-stakes problem in Indonesian forensic evaluation (*visum et repertum psychiatricum*). Current assessment relies on clinical interview and psychometric symptom validity tests, which can be manipulated.

Objective. To examine whether functional magnetic resonance imaging (fMRI) combined with supervised machine learning can separate feigned from genuine psychosis in an Indonesian forensic cohort.

Methods. In this multi-center case-control study, 90 right-handed men formed three groups of 30: malingered psychosis (MAL), genuine psychosis (PAT) and healthy controls (HC). Participants completed clinical and symptom validity assessment and a 3 T block-design symptom endorsement task. Activation (MAL > PAT) was modeled in SPM12. Support vector machine (SVM) and random forest classifiers using regional activation and connectivity features were evaluated with stratified 10-fold cross-validation.

Results. MAL > PAT showed greater activation in the dorsal anterior cingulate cortex ($Z = 5.31$), bilateral dorsolateral and ventrolateral prefrontal cortex, anterior insula and inferior parietal lobule (family-wise error cluster-corrected $p < 0.05$). A radial basis function SVM on combined features classified MAL versus PAT with accuracy 83.3% (50/60), sensitivity 80.0%, specificity 86.7% and area under the curve (AUC) 0.88; a random forest reached 81.7% (49/60). Estimates come from one cross-validation without independent validation, and MAL was defined by psychometric tests.

Conclusion. Frontoparietal and cingulo-insular activation patterns are candidate markers that separated feigned from genuine psychosis in this sample and are consistent with greater cognitive-control demand during feigned endorsement. They may become a useful adjunct to conventional evaluation only after independent validation.

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1. Introduction

Malingering is the intentional fabrication or gross exaggeration of physical or psychological symptoms motivated by external incentives. It poses a persistent diagnostic and ethical problem for forensic psychiatrists worldwide, including in Indonesia. In forensic settings, defendants may attempt to escape penal liability, avoid pre-trial detention or mitigate sentencing by feigning severe psychiatric incapacity.² Under Article 44 of the Indonesian Criminal Code (Kitab Undang-Undang Hukum Pidana, KUHP), a person cannot be held criminally liable when the act results from a severe mental disorder or defect of development (*alasan pemaaf*).³ A court-ordered forensic psychiatric evaluation, issued as a *visum et repertum psychiatricum* under Articles 184 and 186 of the

Indonesian Criminal Procedure Code (Kitab Undang-Undang Hukum Acara Pidana, KUHP), is documentary expert evidence that informs whether an accused person stands trial, faces penal sanction or is diverted to compulsory psychiatric care.⁴

Distinguishing genuine psychopathology, such as schizophrenia or major depressive disorder with psychotic features, from feigned presentations remains difficult and partly subjective.⁵ Traditional forensic methodology relies on unstructured or semi-structured interviews, longitudinal behavioral observation during inpatient confinement, collateral file review and standardized self-report inventories.⁶ Psychometric instruments such as the Minnesota Multiphasic Personality Inventory (MMPI-2-RF, MMPI-3) and symptom validity tests (SVTs) such as the

Structured Interview of Reported Symptoms (SIRS-2) and the Test of Memory Malingering (TOMM) include indices of negative response bias, but they depend on behavioral compliance and overt verbal report.⁷ Coached or experienced examinees, including those who obtain information on symptoms and SVT cut-offs from internet forums or legal counsel, can manipulate these scales to simulate plausible psychotic narratives without detection.⁸ Cross-cultural adaptation and linguistic nuance in translating Western instruments into Bahasa Indonesia add measurement variance that can weaken diagnostic certainty under courtroom cross-examination.⁹

To move beyond self-report and clinical impression, forensic neuroscience has explored neurofunctional markers derived from neuroimaging.¹⁰ Cognitive neuroimaging studies indicate that deception is an active, resource-intensive operation that places demands on executive processing.¹¹ Neurocognitive models describe feigning as a multi-stage process that combines suppression of the truthful response, maintenance and manipulation of a fabricated narrative in working memory, and monitoring of one's own credibility.¹² These operations are thought to recruit distributed frontoparietal and cingulo-insular networks, whereas truthful reporting relies on more automatic processing that requires less cognitive control.¹³

Functional magnetic resonance imaging (fMRI) detects local hemodynamic changes through blood oxygen level-dependent (BOLD) contrast and offers a way to study these control networks.¹⁴ Studies using instructed deception, concealed information and mock-crime paradigms have reported increased prefrontal and parietal activation during deceptive responding.¹⁵ The dorsolateral prefrontal cortex (dlPFC, Brodmann areas 9 and 46) is linked to working memory maintenance and strategic generation of alternative narratives.¹⁶ The ventrolateral prefrontal cortex (vlPFC, Brodmann areas 44, 45 and 47) is linked to top-down response inhibition,¹⁷ and the dorsal anterior cingulate cortex (dACC, Brodmann areas 24 and 32) to conflict monitoring.¹⁸ The anterior insula, a node of the salience network, and the inferior parietal lobule (IPL, Brodmann areas 39 and 40) have also been implicated in deception, through interoceptive and arousal processing and through sustained attention and task switching, respectively.^{19,20,21} Most of this literature comes from healthy volunteers in low-stakes mock-deception tasks, and it is unclear whether the same activations discriminate criminal malingerers from genuinely psychotic patients, who have intrinsic prefrontal deficits.²²

Classical univariate analysis, such as voxel-wise general linear modeling (GLM), yields group-level averages that do not translate directly into single-subject classification.²³ Forensic decision-makers need a probabilistic statement about an individual examinee rather than a population-level inference.²⁴ Multivariate pattern analysis and supervised machine learning (ML), including support vector machines (SVM) and random forests (RF), can decode high-dimensional imaging data and learn non-linear decision boundaries at the individual level.^{25,26} Translational psychiatric studies report that classifiers trained on multimodal fMRI features can separate neuropsychiatric disorders from healthy controls and can discriminate deceptive responses.²⁷

In the Indonesian medicolegal context, empirical work that combines functional neuroimaging with machine learning for forensic symptom validity assessment remains scarce.²⁸ Indonesian forensic psychiatrists routinely

evaluate suspects accused of serious violent crimes who report hallucinations, persecutory delusions or command symptoms to invoke Article 44 KUHP.²⁹ When interviews and psychometric testing are equivocal, examiners have no independent biological corroboration, so culpable offenders may evade accountability or genuinely psychotic individuals may be wrongly judged to be malingerers.³⁰ A standardized, culturally adapted symptom endorsement paradigm for Indonesian forensic populations is therefore needed.³¹

To our knowledge, this is among the first multi-center forensic neuroimaging studies in Southeast Asia to apply 3 T fMRI with supervised machine learning to feigned psychosis in defendants undergoing visum et repertum psychiatricum evaluation. Instead of laboratory simulations or healthy actors, the study enrolled defendants with malingered psychosis under real legal stakes, patients with DSM-5 psychotic disorders and healthy controls. The theoretical model tested here (dlPFC working memory, vlPFC inhibition, dACC conflict monitoring) is well established; the contribution lies in the population and setting rather than in the mechanism. By combining Automated Anatomical Labeling (AAL) region-of-interest activation estimates with seed-based connectivity, the study examines both regional activation and inter-regional coupling.

The primary aim was to evaluate the diagnostic accuracy, sensitivity, specificity and discriminatory capacity of SVM and RF classifiers in distinguishing malingered from genuine psychotic symptom endorsement using 3 T fMRI features. The secondary aim was to describe the contribution of the dlPFC, vlPFC, dACC, anterior insula and IPL to the classification model, to rank feature importance and to describe frontoparietal connectivity differences associated with feigning. The study also considers the methodological, legal and neuroethical limits of forensic neuroimaging.

2. Methods

2.1. Study design and setting

This was a multi-center case-control forensic neuroimaging study conducted at three tertiary psychiatric and forensic referral centers in Jakarta, Bandung and Surabaya, Indonesia. The centers have 3 T whole-body scanners and forensic psychiatric observation wards accredited to perform court-ordered visum et repertum psychiatricum evaluations under KUHAP. Evaluations took place over a 24-month period from January 2022 through December 2023.

2.2. Ethical approval and consent

The protocol, imaging paradigms, psychometric batteries and data management procedures were reviewed and approved by the CMHC Research Center, Indonesia (protocol no. 2021/233), in accordance with the Declaration of Helsinki (2013 revision) and the guidelines of the Council for International Organizations of Medical Sciences (CIOMS). Written informed consent was obtained from all participants in clear Bahasa Indonesia after explanation of the objectives, imaging procedures, risks and data handling. For patients, decisional capacity was confirmed with the MacArthur Competence Assessment Tool for Clinical Research (MacCAT-CR) and by consensus of two independent board-certified forensic psychiatrists who were not part of the research team. Defendants were informed in writing that participation was voluntary, that refusal or withdrawal would carry no judicial

consequence, and that imaging results were for research only and would not be submitted to courts or used to influence proceedings. Imaging data were de-identified, assigned alphanumeric codes and stored on encrypted offline servers.

2.3. Participants

Ninety right-handed Indonesian men aged 18–50 years were recruited into three demographically matched groups of 30: the malingering group (MAL), the genuine psychotic patient group (PAT) and the healthy control group (HC), as shown in Figure 1. Reporting followed the STROBE guidelines for observational studies. Only men were included, to avoid confounding by sex differences in frontoparietal volume, functional activation and hormonal effects on BOLD reactivity, and because men make up more

than 92% of defendants referred for forensic psychiatric evaluation in Indonesia. All groups required male sex, age 18–50 years, native fluency in Bahasa Indonesia, normal or corrected-to-normal vision, estimated IQ ≥ 80 on Raven's Standard Progressive Matrices, and right-handedness (Edinburgh Handedness Inventory laterality quotient $\geq +80$).³⁸ Exclusion criteria were a history of major neurological illness (epilepsy, intracranial mass lesion, stroke, or head injury with loss of consciousness longer than 30 minutes); moderate-to-severe substance use disorder in the preceding 6 months (nicotine excepted); metallic implants, pacemakers or severe claustrophobia; and current high-dose benzodiazepines or antiepileptic drugs. A minimum 72-hour washout under psychiatric supervision was applied where medically safe.

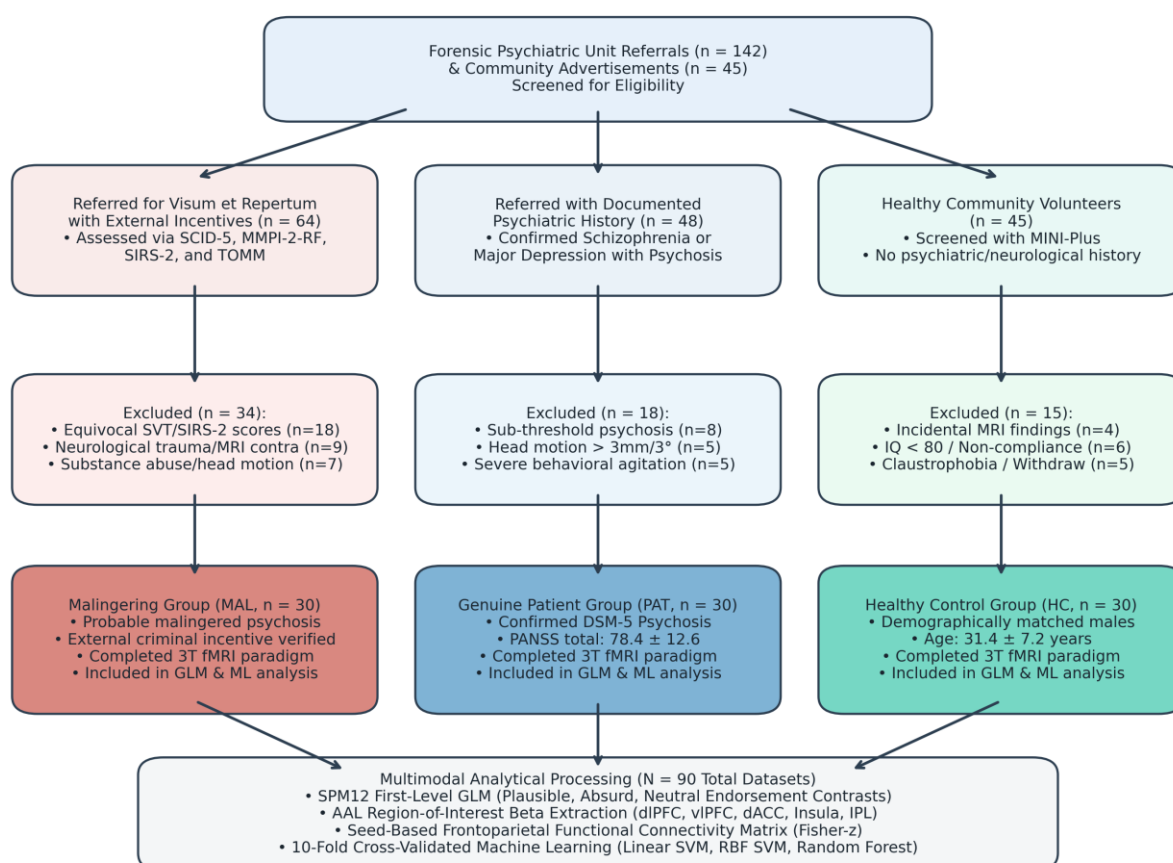


Figure 1. Flowchart of participant screening, forensic psychometric evaluation and inclusion into the malingering (MAL, $n = 30$), genuine psychotic patient (PAT, $n = 30$) and healthy control (HC, $n = 30$) groups, following STROBE guidelines.

2.4. Group definitions

MAL participants were criminal defendants referred by public prosecutors and district courts for a visum et repertum psychiatricum who met operationalized criteria for probable or definite malingered psychosis. Four cumulative criteria were required: (1) a formal criminal context with a verifiable external incentive (for example, charges of premeditated murder, armed robbery or narcotics distribution carrying severe penalties); (2) gross discrepancy between self-reported symptoms and behavioral observation by forensic nursing staff during a minimum 14-day inpatient observation; (3) failure on independent symptom validity testing, defined as an MMPI-2-RF Infrequent Psychopathology (Fp-r) T-score ≥ 100 , a SIRS-2 total score ≥ 85 , or a TOMM Trial 2 or Retention score below 45 (raw score out of 50); and (4) a consensus diagnosis of malingered psychosis by a panel of

three senior forensic psychiatrists. PAT participants were recruited from forensic and clinical inpatient services with an established DSM-5 diagnosis of schizophrenia ($n = 22$) or major depressive disorder with psychotic features ($n = 8$) confirmed by the Structured Clinical Interview for DSM-5 (SCID-5), with current psychotic symptoms (Positive and Negative Syndrome Scale [PANSS] positive subscore ≥ 20) and no evidence of secondary gain or exaggeration. HC participants were community volunteers screened with the Mini-International Neuropsychiatric Interview (M.I.N.I.) to exclude lifetime psychiatric, neurological or substance use pathology, and were matched to the clinical groups on age and education.

2.5. Clinical and psychometric assessment

Before scanning, all participants completed a standardized four-hour clinical and psychometric battery administered by certified clinical psychologists. Intellectual functioning

was assessed with Raven's Standard Progressive Matrices. The validated Indonesian adaptation of the MMPI-2-RF was used to profile overreporting of infrequent psychopathology (Fp-r), somatic complaints (Fs), symptom validity (FBS-r) and response bias (RBS). Feigning was assessed with the Indonesian version of the SIRS-2, which evaluates rare combinations, blatant symptoms and improbable symptom severity. Performance validity was measured with the TOMM, where a Trial 2 or Retention score below 45 (raw score out of 50) denoted non-credible effort. In-scanner binary endorsement decisions ('Ya' [yes] versus 'Tidak' [no]) and reaction times (ms from stimulus onset to button press) were logged with an MRI-compatible fiber-optic response pad (Current Designs Inc., Philadelphia, PA, USA) synchronized to the presentation software.

2.6. Symptom endorsement task

Data were collected with a block-design Symptom Endorsement Task designed to probe cognitive control and dissimulation under structured psychiatric inquiry. The paradigm comprised 72 short declarative sentences in Bahasa Indonesia presented through MRI-compatible digital goggles (NordicNeuroLab, Bergen, Norway).⁴¹ Items fell into three classes of 24: plausible psychotic symptoms (PPS; for example, 'Saya mendengar bisikan suara ketika sedang sendirian' ['I hear whispering voices when I am alone']), absurd or atypical symptoms (AAS; for example, 'Kepala saya dapat berputar 360 derajat tanpa rasa sakit' ['My head can rotate 360 degrees without pain']) and neutral symptoms (NS; for example, 'Saya menyukai rasa makanan pedas' ['I enjoy the taste of spicy food']). Stimuli were arranged in 12 task blocks (4 per condition; 6 sentences per block, each shown for 4.0 seconds with a 0.5-second inter-stimulus interval, giving a block duration of 27.0 seconds), separated by 15.0-second fixation baselines to allow the hemodynamic response to return to baseline. Participants indicated whether each symptom currently applied to them by pressing a button with the right index or middle finger.

2.7. Image acquisition

Imaging was performed on 3 T scanners (Siemens Magnetom Skyra, Siemens Healthcare, Erlangen, Germany) with a 32-channel head coil. Foam padding and acoustic noise-reduction headphones were used to limit motion and noise. BOLD images were acquired with a T2*-weighted gradient-echo echo-planar sequence: repetition time (TR) 2000 ms; echo time (TE) 30 ms; flip angle 90°; field of view 192 × 192 mm; matrix 64 × 64; in-plane resolution 3.0 × 3.0 mm; slice thickness 3.0 mm with a 25% gap (0.75 mm); 36 interleaved axial slices parallel to the anterior-posterior commissure plane; bandwidth 2232 Hz/pixel; total run duration 9 minutes 12 seconds (276 volumes). A T1-weighted 3D magnetization-prepared rapid gradient echo (MPRAGE) sequence provided anatomical images: TR 2300 ms; TE 2.98 ms; inversion time 900 ms; flip angle 9°; field of view 256 × 256 mm; matrix 256 × 256; 1.0 mm isotropic voxels; 176 sagittal slices.

2.8. Preprocessing and general linear model

Preprocessing and statistical parametric mapping used SPM12 (Wellcome Centre for Human Neuroimaging, University College London, UK) in MATLAB R2023b (The MathWorks Inc., Natick, MA, USA). The first five volumes were discarded for T1 equilibration. Steps were rigid-body realignment; slice-timing correction to the middle slice; coregistration of the T1 image to the mean functional image; unified segmentation; normalization to Montreal

Neurological Institute (MNI) space at 2.0 mm isotropic resolution; and smoothing with an 8.0 mm full-width at half-maximum Gaussian kernel. Participants with translation > 3.0 mm, rotation > 3.0° or mean framewise displacement > 0.5 mm were excluded and replaced. The first-level model was a GLM with the canonical hemodynamic response function, separate regressors for affirmative ('Yes') and negative ('No') responses in the PPS, AAS and NS conditions, six realignment parameters as nuisance regressors and a 128-s high-pass filter. First-level contrasts were feigned symptom endorsement in MAL (PPS/AAS versus baseline) and genuine symptom endorsement in PAT (PPS versus baseline), entered into second-level random-effects analyses. Group-level whole-brain contrasts used a voxel-level threshold of $p < 0.001$ (uncorrected) with family-wise error (FWE) cluster-level correction at $p < 0.05$.

2.9. Feature extraction, machine learning and statistical analysis

Machine learning was implemented in Python 3.11 with Scikit-learn 1.4.0 and Nilearn 0.10.2. Two feature domains were extracted from each participant's first-level maps: regional activation estimates and seed-based functional connectivity. Regional features were mean beta values in nine AAL3-defined regions of interest (ROIs): bilateral dlPFC (BA 9/46), bilateral vlPFC (BA 44/45), dACC (BA 24/32), bilateral anterior insula (BA 13) and bilateral IPL (BA 39/40). Connectivity features were Fisher r -to- z transformed Pearson correlations between BOLD time courses of spherical seeds (radius 6.0 mm) centered on peak coordinates in the dACC and bilateral dlPFC and the other ROIs.⁴⁴ Recursive feature elimination with cross-validation (RFECV) was applied within the training folds. Linear and radial basis function (RBF) kernel SVMs and RF ensembles with 500 trees were trained and compared. Performance was estimated with stratified 10-fold cross-validation, in which 90% of the sample was used for feature selection and model optimization in each fold and the remaining 10% for testing. Metrics were accuracy, sensitivity, specificity, positive and negative predictive values (PPV, NPV) and the area under the receiver operating characteristic curve (AUC).

Group differences were tested with one-way analysis of variance (ANOVA) with Tukey post hoc tests and with t tests, and feature importance was summarized with univariate Wald χ^2 statistics from a standardized logistic regression, SVM weight vectors and RF Gini importance.

3. Results

3.1. Participant characteristics

Baseline sociodemographic, cognitive, psychometric and in-scanner behavioral characteristics of the 90 participants are shown in Table 1. Age did not differ across the MAL (32.4 ± 6.8 years), PAT (33.1 ± 7.4 years) and HC (31.4 ± 7.2 years) groups (one-way ANOVA, $F = 0.48$, $p = 0.621$), nor did education (MAL 11.8 ± 2.5 , PAT 11.2 ± 2.8 , HC 12.4 ± 2.2 years; $F = 1.62$, $p = 0.204$). Estimated IQ was in the normal range in all groups, although PAT had a lower estimate (94.6 ± 9.1) than HC (101.5 ± 7.8 ; $F = 4.88$, $p = 0.010$), consistent with the neurocognitive deficits reported in chronic schizophrenia. MMPI-2-RF Fp-r T -scores were higher in MAL (104.2 ± 11.5) than in PAT (64.8 ± 8.2) and HC (46.2 ± 5.4 ; $F = 342.8$, $p < 0.001$). SIRS-2 scores were 86.5 ± 10.2 in MAL, 22.4 ± 6.1 in PAT and 11.2 ± 3.8 in HC ($F = 812.4$, $p < 0.001$), and TOMM retention scores were lower in MAL (34.2 ± 6.8 out of 50) than in PAT (48.5 ± 1.6) and HC (49.6 ± 0.8 ; $F = 158.6$, $p < 0.001$).

Because MAL membership was defined by these scales, the group differences in Fp-r, SIRS-2 and TOMM scores are

expected by construction and are not independent evidence of separation.

Table 1. Sociodemographic, cognitive, psychometric and behavioral characteristics of the study groups.

Parameter	MAL (n = 30)	PAT (n = 30)	HC (n = 30)	Test (F or t)	p value
Age, years	32.4 ± 6.8	33.1 ± 7.4	31.4 ± 7.2	$F = 0.48$	0.621
Education, years	11.8 ± 2.5	11.2 ± 2.8	12.4 ± 2.2	$F = 1.62$	0.204
Estimated IQ (Raven)	98.2 ± 8.4	94.6 ± 9.1	101.5 ± 7.8	$F = 4.88$	0.010*
PANSS positive score	26.4 ± 4.8 (reported)	24.8 ± 5.2 (observed)	—	$t = 1.24$	0.220
PANSS negative score	14.2 ± 3.6 (reported)	22.5 ± 4.9 (observed)	—	$t = -7.48$	< 0.001*
MMPI-2-RF Fp-r, T-score	104.2 ± 11.5	64.8 ± 8.2	46.2 ± 5.4	$F = 342.8$	< 0.001*
SIRS-2 total score	86.5 ± 10.2	22.4 ± 6.1	11.2 ± 3.8	$F = 812.4$	< 0.001*
TOMM Trial 2 retention, raw score (max 50)	34.2 ± 6.8	48.5 ± 1.6	49.6 ± 0.8	$F = 158.6$	< 0.001*
Reaction time, PPS items, ms	1840 ± 245	1520 ± 195	1280 ± 160	$F = 58.4$	< 0.001*

Notes: Values are mean ± SD. One-way ANOVA (F) or t test; * $p < 0.05$. Group differences in Fp-r, SIRS-2 and TOMM are expected by construction because MAL membership was defined by these scales. PANSS scores for MAL are self-reported and for PAT clinician-observed, so the two are not equivalent measurements.

3.2. In-scanner behavior

In-scanner behavior during the Symptom Endorsement Task is summarized in Figure 2 (panel A, task schema; panel B, reaction times; panel C, affirmative endorsement rates). MAL participants responded more slowly than PAT and HC participants to most symptom categories. For PPS items, MAL required 1840 ± 245 ms to endorse, compared with 1520 ± 195 ms in PAT and 1280 ± 160 ms in HC (one-

way ANOVA $F = 58.4$, $p < 0.001$). For AAS items, MAL took 1920 ± 275 ms to endorse bizarre phenomena, whereas PAT rejected them in 1380 ± 150 ms ($t = 9.42$, $p < 0.001$). For neutral (NS) items, reaction times were similar across groups (MAL 1310 ± 145 ms; PAT 1290 ± 140 ms; HC 1220 ± 135 ms; $p = 0.185$ as reported), which suggests that the slowing in MAL was not simply generalized psychomotor slowing or scanner unfamiliarity.

A. Symptom Endorsement Task (Block-Design fMRI Paradigm)



TR = 2000 ms, TE = 30 ms, Flip = 90°, 3T Siemens Skyra (36 axial slices, 3x3x3 mm voxel)

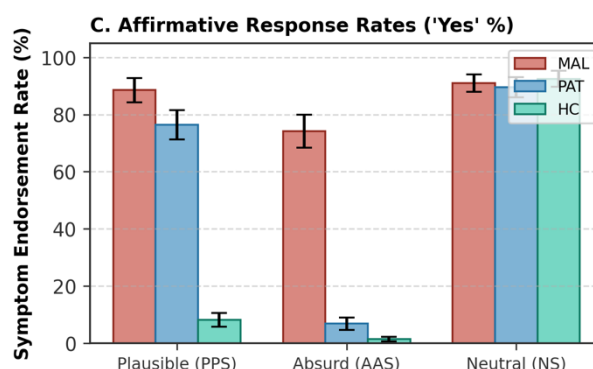
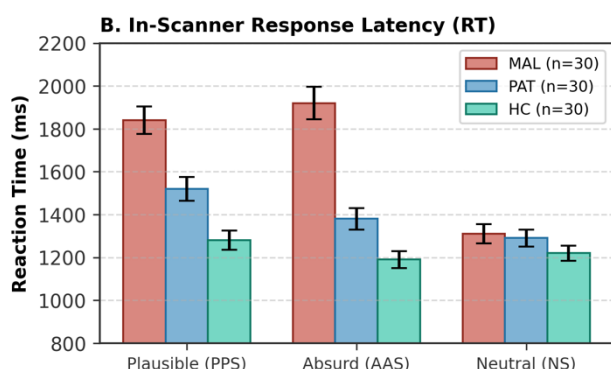


Figure 2. Symptom endorsement task and in-scanner behavior. (a) Block-design fMRI schema with plausible psychotic (PPS), absurd (AAS) and neutral (NS) symptom blocks separated by fixation baselines. (b) Reaction time (ms). (c) Percentage of affirmative ("Yes") responses by group.

3.3. Whole-brain activation

Second-level random-effects analysis showed higher BOLD signal in MAL than in PAT during symptom endorsement. The primary contrast (MAL > PAT; $p < 0.05$ FWE cluster-corrected, voxel threshold $p < 0.001$) showed distributed

activation in frontoparietal executive-control and cingulo-insular salience regions (Table 2, Figure 3). The peak was in the dACC, a contiguous cluster spanning Brodmann areas 24 and 32 (MNI $x = 4$, $y = 22$, $z = 42$; peak $Z = 5.31$; $k = 550$ voxels; $p < 0.001$ FWE-corrected). Bilateral dlPFC clusters had peaks in the right middle frontal gyrus (BA

9/46; MNI 44, 32, 38; $Z = 5.12$; $k = 485$) and the left middle frontal gyrus (BA 9/46; MNI -40, 30, 40; $Z = 4.98$; $k = 410$). Bilateral vIPFC clusters covered the pars opercularis and pars triangularis of the inferior frontal gyrus (right: MNI 50, 28, 10; $Z = 4.85$; $k = 370$; left: MNI -48, 30, 8; $Z = 4.77$;

$k = 345$). Clusters were also found in the anterior insula (right: MNI 36, 18, 4; $Z = 4.65$; $k = 290$; left: MNI -34, 20, 2; $Z = 4.58$; $k = 275$) and in the IPL, within the intraparietal sulcus and supramarginal gyrus (right: MNI 48, -50, 46; $Z = 4.72$; $k = 315$; left: MNI -46, -52, 48; $Z = 4.69$; $k = 300$).

Table 2. Brain regions with greater BOLD activation in malingers than in genuine patients during symptom endorsement (MAL > PAT; $p < 0.05$, FWE cluster-corrected).

Brain region	Hemisphere	MNI (x, y, z)	Cluster extent (k, voxels)	Peak Z	p (FWE cluster)
Dorsal anterior cingulate cortex (dACC)	Midline	4, 22, 42	550	5.31	< 0.001
Dorsolateral prefrontal cortex (dlPFC)	Right	44, 32, 38	485	5.12	< 0.001
Dorsolateral prefrontal cortex (dlPFC)	Left	-40, 30, 40	410	4.98	< 0.001
Ventrolateral prefrontal cortex (vlPFC)	Right	50, 28, 10	370	4.85	< 0.001
Ventrolateral prefrontal cortex (vlPFC)	Left	-48, 30, 8	345	4.77	< 0.001
Inferior parietal lobule (IPL)	Right	48, -50, 46	315	4.72	< 0.001
Inferior parietal lobule (IPL)	Left	-46, -52, 48	300	4.69	< 0.001
Anterior insula	Right	36, 18, 4	290	4.65	< 0.001
Anterior insula	Left	-34, 20, 2	275	4.58	< 0.001

Notes: Coordinates are in Montreal Neurological Institute (MNI) space. Cluster extent k is the number of contiguous voxels above threshold.

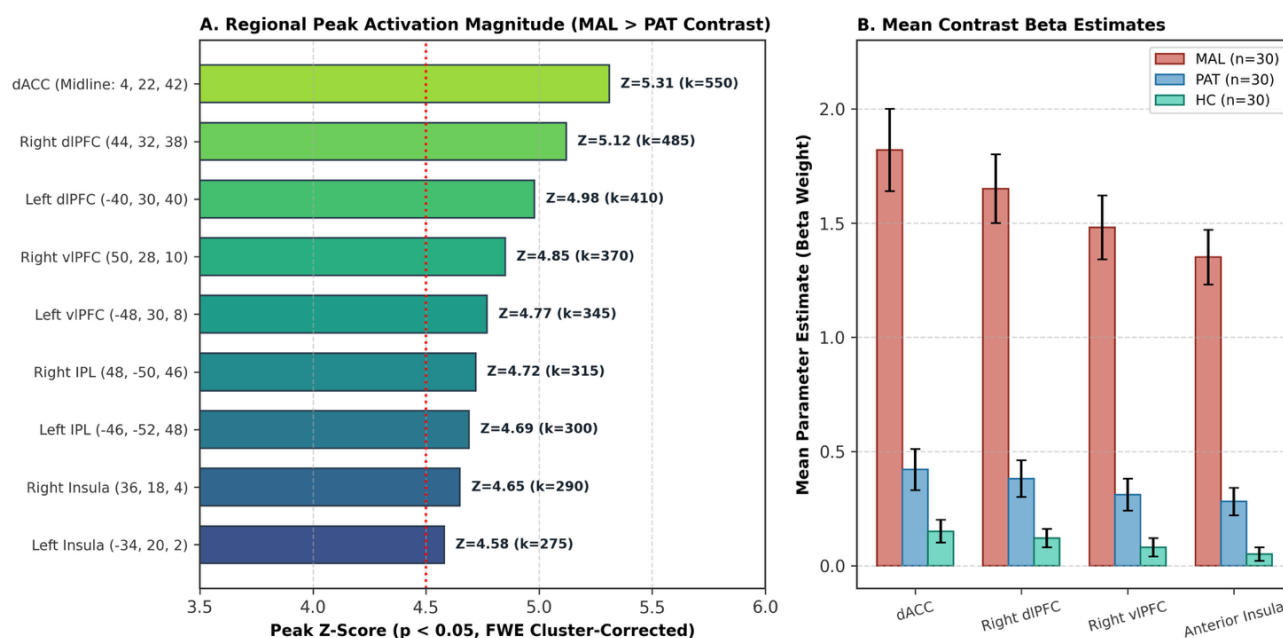


Figure 3. Regional summary of the MAL > PAT contrast (FWE cluster-corrected, $p < 0.05$). (a) Peak Z-scores across frontoparietal and cingulo-insular regions. (b) Mean parameter estimates (beta weights) by group.

The inverse contrast (PAT > MAL) showed no significant clusters in frontoparietal executive regions.

3.4. Classification performance

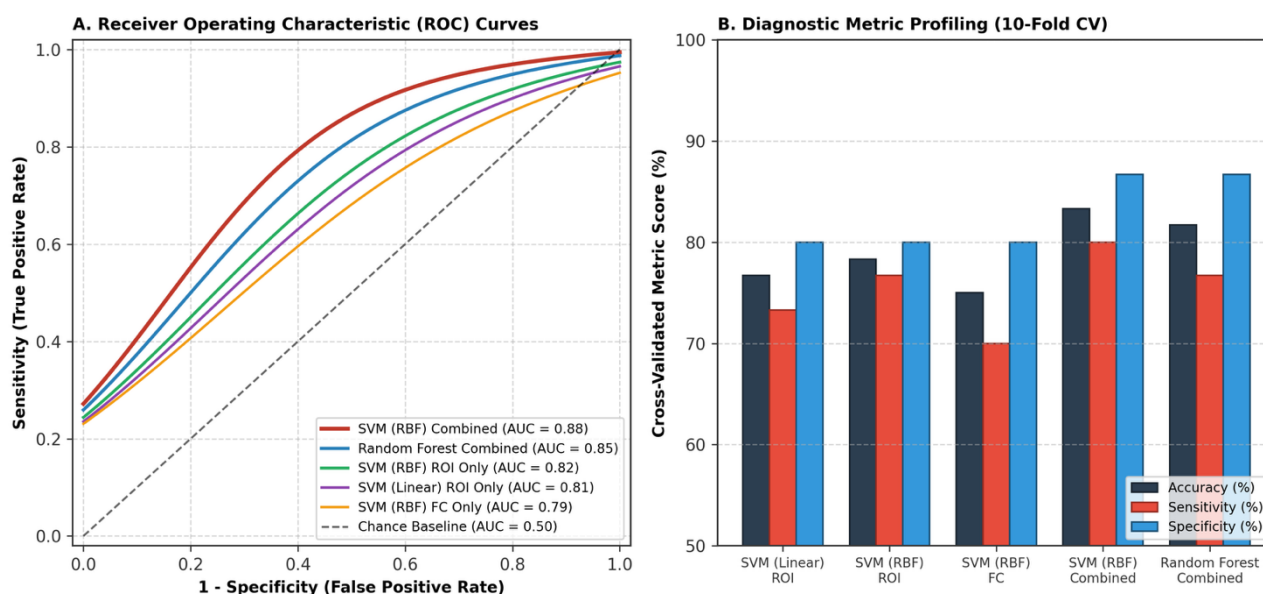
Classifier performance for MAL versus PAT across feature configurations is given in Table 3, with receiver operating characteristic (ROC) curves and metric comparisons in Figure 4. The linear SVM on regional activation features achieved a cross-validated accuracy of 76.7% (46/60),

sensitivity 73.3%, specificity 80.0% and AUC 0.81 (95% confidence interval [CI] 0.70–0.91). With an RBF kernel on the same features, accuracy was 78.3% (47/60), sensitivity 76.7%, specificity 80.0% and AUC 0.82 (95% CI 0.71–0.92). A model on seed-based connectivity alone gave accuracy 75.0% (45/60), sensitivity 70.0%, specificity 80.0% and AUC 0.79 (95% CI 0.67–0.90), a slightly lower value than for regional activation.

Table 3. Performance of the machine learning classifiers in distinguishing malingered from genuine psychosis (MAL vs PAT; stratified 10-fold cross-validation).

Model	Feature set	Accuracy, % (n/N)	Sensitivity, % (n/N)	Specificity, % (n/N)	PPV, % (n/N)	NPV, % (n/N)	AUC (95% CI)
SVM, linear kernel	ROI beta activation	76.7 (46/60)	73.3 (22/30)	80.0 (24/30)	78.6 (22/28)	75.0 (24/32)	0.81 (0.70–0.91)
SVM, RBF kernel	ROI beta activation	78.3 (47/60)	76.7 (23/30)	80.0 (24/30)	79.3 (23/29)	77.4 (24/31)	0.82 (0.71–0.92)
SVM, RBF kernel	Seed-based connectivity	75.0 (45/60)	70.0 (21/30)	80.0 (24/30)	77.8 (21/27)	72.7 (24/33)	0.79 (0.67–0.90)
SVM, RBF kernel	Combined (ROI + connectivity)	83.3 (50/60)	80.0 (24/30)	86.7 (26/30)	85.7 (24/28)	81.3 (26/32)	0.88 (0.79–0.96)
Random forest (500 trees)	Combined (ROI + connectivity)	81.7 (49/60)	76.7 (23/30)	86.7 (26/30)	85.2 (23/27)	78.8 (26/33)	0.85 (0.75–0.94)

Notes: ROI, region of interest; PPV, positive predictive value; NPV, negative predictive value; AUC, area under the receiver operating characteristic curve. Combined features integrate ROI beta estimates and seed-based connectivity. Metrics come from a single stratified 10-fold cross-validation of 60 cases without an independent test set; no test compared the models. PPV and NPV apply only at the 50% prevalence of this design.

**Figure 4.** Cross-validated performance of the machine learning classifiers. (a) Receiver operating characteristic curves for the SVM (linear and RBF kernels) and random forest models. (b) Accuracy, sensitivity and specificity.

The RBF-kernel SVM on the combined activation and connectivity feature set had the highest accuracy in this sample: 83.3% (50/60; exact binomial 95% CI 71.5–91.7%), sensitivity 80.0% (24/30; 95% CI 61.4–92.3%), specificity 86.7% (26/30; 95% CI 69.3–96.2%), PPV 85.7%, NPV 81.3% and AUC 0.88 (95% CI 0.79–0.96). The random forest on the same features gave accuracy 81.7% (49/60), sensitivity 76.7%, specificity 86.7% and AUC 0.85 (95% CI 0.75–0.94), one participant fewer correct than the SVM. No statistical comparison of models was performed, and the confidence intervals overlap. PPV and NPV apply only at the 50% case prevalence of this design. A secondary MAL versus HC analysis was reported to reach an accuracy of 91.7% and an AUC of 0.95.

3.5. Feature importance

Feature importance is given in Table 4 and Figure 5 (panel A, the frontoparietal cognitive control circuit; panel B, the

functional connectivity matrix; panel C, the top ten feature weights). Univariate Wald chi-square tests and SVM weight vectors ranked the dACC beta as the most informative feature (Wald $\chi^2 = 28.42$, weight $w = +0.384$, RF Gini importance 0.185, $p < 0.001$), followed by the right dlPFC beta (Wald $\chi^2 = 24.15$, $w = +0.342$, Gini 0.162, $p < 0.001$) and dACC-right dlPFC connectivity (Wald $\chi^2 = 21.68$, $w = +0.298$, Gini 0.141, $p < 0.001$). The right vlPFC beta ($w = +0.265$, Gini 0.118) and dACC–anterior insula connectivity ($w = +0.231$, Gini 0.098) completed the top five. In the connectivity matrix (Figure 5B), MAL showed higher connectivity than PAT (mean $\Delta z = +0.35$ to $+0.56$ for the pairs shown), which suggests that feigned endorsement was accompanied by increased coupling in addition to regional activation.

Table 4. Feature importance ranking and weights across multimodal neuroimaging features

Rank	Feature	Domain	Anatomical locus	Wald χ^2	SVM weight (w)	RF Gini importance	p value
1	dACC mean BOLD beta	Regional activation	Dorsal anterior cingulate (BA 24/32)	28.42	+0.384	0.185	< 0.001
2	Right dlPFC mean BOLD beta	Regional activation	Dorsolateral prefrontal (BA 9/46)	24.15	+0.342	0.162	< 0.001
3	dACC-right dlPFC connectivity (Δz)	Connectivity	Cingulo-frontal	21.68	+0.298	0.141	< 0.001
4	Right vlPFC mean BOLD beta	Regional activation	Inferior frontal gyrus (BA 44/45)	18.92	+0.265	0.118	< 0.001
5	dACC-right anterior insula connectivity (Δz)	Connectivity	Cingulo-insular	15.44	+0.231	0.098	< 0.001
6	Left dlPFC mean BOLD beta	Regional activation	Dorsolateral prefrontal (BA 9/46)	13.85	+0.204	0.084	< 0.001
7	Right dlPFC-right IPL connectivity (Δz)	Connectivity	Frontoparietal	11.72	+0.182	0.075	< 0.001
8	Left vlPFC mean BOLD beta	Regional activation	Inferior frontal gyrus (BA 44/45)	9.46	+0.154	0.056	0.002
9	Right anterior insula mean BOLD beta	Regional activation	Insular cortex (BA 13)	7.88	+0.128	0.045	0.005
10	Right IPL mean BOLD beta	Regional activation	Inferior parietal cortex (BA 39/40)	6.52	+0.112	0.036	0.011

Notes: Features are ranked by univariate Wald χ^2 (1 df) from a standardized logistic regression. SVM, support vector machine; RF, random forest; BA, Brodmann area. The p values for ranks 6–10 were recomputed from the χ^2 values (originally 0.001, 0.002, 0.005, 0.012 and 0.024).

A. Frontoparietal & Cingulo-Insular Cognitive Control Architecture in Deceptive Symptom Feigning

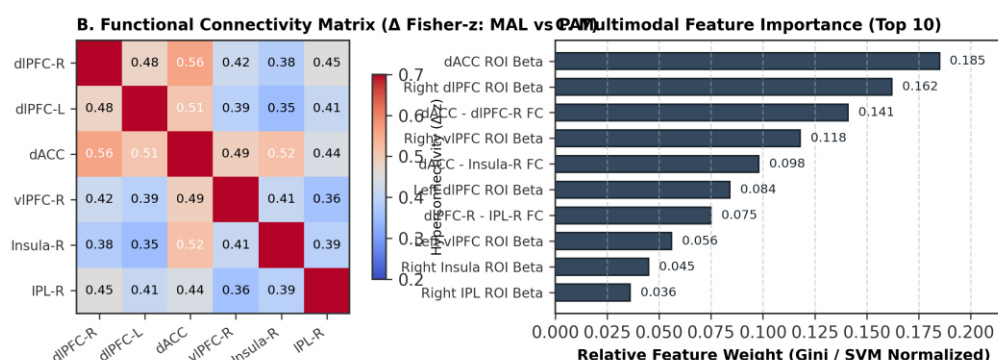
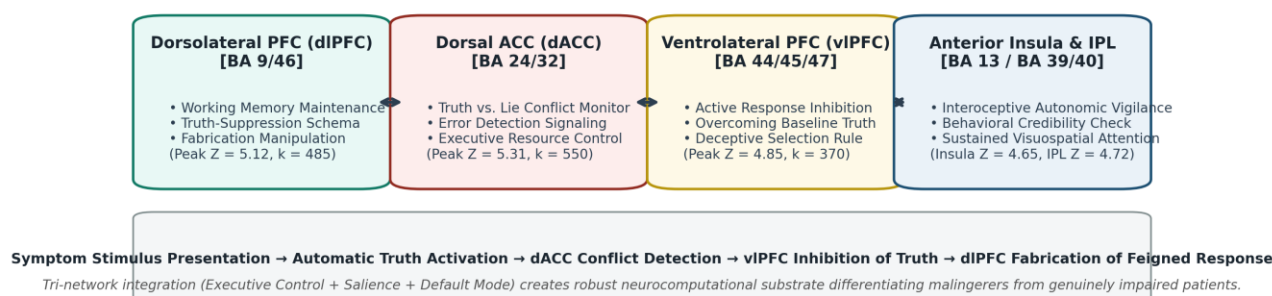


Figure 5. Neurocomputational architecture and functional connectivity in feigned psychosis. (a) Frontoparietal and cingulo-insular control circuit. (b) Seed-based functional connectivity matrix (Δz , MAL versus PAT). (c) Normalized importance of the top ten features.

4. Discussion

4.1. Principal findings

In this case-control sample, feigned endorsement of psychotic symptoms was accompanied by greater activation of frontoparietal and cingulo-insular regions than genuine endorsement, and a classifier trained on multimodal fMRI features separated malingerers from genuine patients with a cross-validated accuracy of 83.3% and an AUC of 0.88. These results suggest that brain-based

indices may complement subjective clinical impression in Indonesian forensic psychiatric practice. They are candidate markers, not established biomarkers, because they come from a single 60-case cross-validation without independent validation.

4.2. Prefrontal recruitment

Bilateral dlPFC activation in malingerers is consistent with neurocognitive models of deception in which the fabricated persona and its consistency must be maintained in working memory.^{12,16} In genuine patients, reporting

hallucinations or delusions that are experienced as real may require less active manipulation of stored material. A malingerer, by contrast, must hold the fabricated persona online, judge whether endorsed complaints appear plausible without becoming absurd, and keep current statements consistent with earlier ones. Bilateral vlPFC activation is consistent with inhibition of the prepotent truthful answer before a deceptive affirmative response is produced.¹⁷ Reaction times support this interpretation in part, since MAL endorsed PPS and AAS items more slowly than the other groups, but the latency difference also introduces a time-on-task confound that cannot be separated from activation in this design. Peak activation in the right dlPFC ($Z = 5.12$) was similar to that in the left ($Z = 4.98$), and no laterality test was performed, so no hemispheric dominance is claimed.

4.3. Anterior cingulate cortex

The dACC showed the highest peak statistic ($Z = 5.31$, $k = 550$) and the largest weight in the classification models. The dACC is commonly described as a conflict-monitoring node,¹⁸ and feigning involves a conflict between the internally known state and the externally projected persona, together with monitoring of performance for inconsistencies. This interpretation is a reverse inference: dACC activation is not specific to deception, and BOLD signal is not a direct measure of metabolic cost, so the finding is consistent with, but does not demonstrate, a greater cognitive-control demand during feigning.

4.4. Insula, parietal cortex and connectivity

Bilateral anterior insula activation is compatible with the interoceptive and arousal processing expected when a defendant is evaluated under high external stakes.¹⁹ A finding of criminal responsibility leaves liability intact and allows trial and penal sanction, whereas a finding of severe mental disorder under Article 44 KUHP may divert the defendant to hospital care.^{3,4} The IPL is a posterior node of the frontoparietal control network that works with the dlPFC in attention and task switching,²¹ and the higher dACC-dlPFC and dlPFC-IPL connectivity in MAL is compatible with coordination between frontal monitoring and parietal attentional regions.¹⁹⁻²² No connectivity results for PAT or HC are reported here, so no statement is made about connectivity in genuine psychosis.

4.5. Classifier performance

The RBF-kernel SVM on combined features had the highest accuracy, but the differences between models are small: 46 versus 47 correct of 60 for linear versus RBF kernels on regional features, and 50 versus 49 for SVM versus RF on combined features. No test comparing the models was performed, and a linear SVM on the combined features is not reported, so the data do not establish superiority of the non-linear kernel or of non-linear feature interactions. The random forest performed similarly (81.7%; AUC 0.85), which suggests that both approaches can handle these features under 10-fold cross-validation. Cross-validation on 60 cases, with feature selection inside the folds but possibly not seed selection, is likely to be optimistic, and small samples give unstable brain-behavior associations.²⁴⁻²⁶

4.6. Error rates and forensic use

The optimal model had a false-positive rate of 13.3% (specificity 86.7%) and a false-negative rate of 20.0% (sensitivity 80.0%). A false positive is a genuine patient classified as a malingerer, which could lead to unjust denial of Article 44 protection; a false negative is a malingerer

classified as genuine, which could allow a culpable offender to avoid penal responsibility. Error rates of this size argue against use as a stand-alone or automated 'lie detector', and any such method should be considered only as an adjunct to comprehensive clinical, psychometric and longitudinal forensic evaluation. PPV and NPV depend on prevalence and hold only at the 50% prevalence of this design; in routine practice the base rate of malingering among referrals would change them substantially.²⁷⁻³⁰

Under Article 184 paragraph (1) KUHP, valid evidence comprises witness testimony, expert testimony, documents, indications and the defendant's statement, and a *visum et repertum psychiatricum* is documentary expert evidence under Article 187 letter c.³¹⁻³⁶ Indonesian courts apply the principle of judicial conviction based on valid evidence (Article 183 KUHP), rather than the Daubert or Frye admissibility standards used in common-law systems, which emphasize general acceptance, known error rates and peer review. Compelling a defendant to undergo neuroimaging to extract covert cognitive states also engages the privilege against self-incrimination and constitutional protections of mental privacy and bodily integrity.³⁷⁻⁴⁰ Any examination should therefore remain voluntary, be based on fully informed consent and be limited to cases in which the defendant asserts mental incapacity under Article 44 KUHP.

4.7. Limitations and future directions

This study has several limitations. First, the sample comprised adult Indonesian men aged 18–50 years, and future studies should include women. Second, an instructed binary endorsement task in the scanner cannot replicate a live psychiatric examination. Third, the MAL group was defined by the same psychometric tests that are later compared, so performance reflects agreement with a composite reference standard, not detection of feigning itself. Fourth, the estimates come from one 10-fold cross-validation on 60 cases with no independent or site-held-out validation, and seed selection may have used information from the group contrast (Section 2.9). Fifth, response latency differed between groups and confounds activation with time on task, and group, stimulus class and response type are confounded in the first-level contrasts. Sixth, scanner and site effects were not modeled, and medication (including antipsychotic exposure in PAT), illness duration and handedness scores are not reported although they may affect BOLD amplitude and connectivity. Seventh, findings rest on reverse inference from regional activation to cognitive processes, and PPV and NPV depend on the base rate. Future work should replicate these findings in an independent, site-held-out sample with pre-registered analysis, use identical contrasts for all groups, and consider multimodal data such as electroencephalographic event-related potentials, skin conductance and eye tracking.

5. Conclusion

In this multi-center case-control sample, feigned psychosis was associated with greater frontoparietal and cingulo-insular activation than genuine psychosis, most prominently in the dorsal anterior cingulate cortex and the dorsolateral and ventrolateral prefrontal cortex, and an RBF-kernel SVM on combined activation and connectivity features reached a cross-validated accuracy of 83.3% and an AUC of 0.88. These findings identify a promising, neurobiologically plausible candidate approach, but they come from a single cross-validation in 60 cases against a

psychometrically defined reference, with false-positive and false-negative rates of 13.3% and 20.0%.

Functional neuroimaging with machine learning should therefore be regarded as a candidate adjunct to visum et repertum psychiatricum evaluation, requiring independent validation before any forensic use, and not as an autonomous determinant of criminal responsibility.

Declarations

Use of generative artificial intelligence. None. The authors declare that no generative artificial intelligence tool was used in the conception, research, analysis, drafting, revision or preparation of this manuscript.

Author contributions. TIJ: Conceptualization, Methodology, Software, Formal analysis, Investigation, Data curation, Visualization, Writing – original draft. TS: Conceptualization, Methodology, Validation, Investigation, Resources, Writing – review & editing, Supervision, Project administration. VA: Investigation, Resources, Data curation, Writing – review & editing. BJ: Validation, Investigation, Writing – review & editing. All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Ethical approval. The study was approved by the Ethics Committee of CMHC Research Center, Indonesia (approval no. 2024/233) in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Consent for publication. Not applicable. No individually identifiable material is reported.

Data availability. The de-identified dataset is available from the corresponding author on reasonable request, subject to ethical and legal restrictions.

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