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Family Income Is Associated With Regional Brain Glucose Metabolism in Middle-Aged Males

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ABSTRACT

Socioeconomic status is a multifaceted construct that plays a prominent role in shaping our environment. This study investigated the associations between family income, education level, and brain glucose metabolism in middle-aged males. We retrospectively analysed data of healthy males who underwent a health check-up programme including (1) brain ¹⁸F-fluorodeoxyglucose positron emission tomography, (2) anthropometric measurements, (3) survey of family income and education level and (4) measures of stress, anxiety and depression. Bayesian hierarchical modelling was used to evaluate the relationships between SES variables and regional standardized uptake value ratios (SUVRs). A total of 233 healthy males were included in this study. Family income was positively correlated with education level. Higher family income was associated with increased glucose metabolism in the caudate, putamen, anterior cingulate, hippocampus and amygdala. In contrast, education level showed no significant association with regional brain metabolism in either ROI-based or full-volume analyses. In conclusion, family income, and education level show differential associations with brain glucose metabolism in middle-aged males. Family income is associated with elevated brain glucose metabolism in regions involved in reward processing and stress regulation, suggesting a potential link between current socioeconomic resources and neural activity. However, these findings are cross-sectional and must be interpreted as associative rather than causal. Education level does not show a significant association with brain glucose metabolism.

1 | Introduction

Brain is a social organ (Insel and Fernald 2004). No human brain exists outside of a particular socioeconomic context (Farah 2017). Socioeconomic status (SES) is a multifaceted construct encompassing access to resources like healthcare,

nutrition, educational and occupational opportunities and social standing (Adler et al. 1994). This complex variable is a powerful predictor of a wide array of life outcomes, including both physical and mental health (Farah 2017). The incidence of heart disease, stroke, cancer and diabetes, as well as depression, anxiety and psychosis is positively related to SES (Adler and

Abbreviations: FDG, fluorodeoxyglucose; PET, positron emission tomography; SES, socioeconomic status; SUVR, standardized uptake value ratios.

Kyoungjune Pak and Seunghyeon Shin contributed equally in this work.

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Stewart 2010). Similarly, intelligence and academic achievement exhibit positive gradients with SES (Farah 2017).

The neurobiological pathways where SES exerts these pervasive effects are an active area of investigation. Studies in animal models suggest that social context can induce tangible neurochemical alterations. Morgan et al. demonstrated that in monkeys, social status influenced dopamine receptor (DR) availability; dominant monkeys exhibited increased DR availability after 3 months of social housing compared with subordinate monkeys (Morgan et al. 2002). These neurobiological changes were further linked to differential behavioural responses, such as subordinate monkeys showing increased vulnerability to the reinforcing effects of cocaine, whereas dominant monkeys were found to be resistant to its effects (Morgan et al. 2002). Although these findings in nonhuman primates highlight alterations in neurotransmitter systems due to social hierarchy, further study is needed to understand how broader SES are associated with global brain functional markers such as glucose metabolism in humans.

Examination of the relationship between SES and the brain has primarily focused on the earlier or later stages of the lifespan when the brain is most vulnerable, which are characterized by notable changes in brain structure and function (Hedman et al. 2012). For example, early by the age of 7 months, infant language showed an inverse correlation with lower SES background (Betancourt et al. 2015). Similarly, adolescents from lower SES backgrounds have less access to enriching opportunities and resources, which could shape how adolescents respond to or pursue rewarding experiences (Farah 2018). In the elderly, the relationship between SES and the brain is complicated due to 'cognitive reserve', the ability to maintain cognitive abilities despite pathological changes (Arenaza-Urquijo et al. 2013).

However, a significant gap exists in our understanding of how SES relates to brain function during middle adulthood. This period of life is particularly important as accumulated experiences and exposures associated with SES can have lasting effects on brain health; however, it remains comparatively understudied in the context of SES and direct measures of brain activity. The human brain utilizes glucose as its main source of energy; thus, brain glucose metabolism, assessed by positron emission tomography (PET) with ^{18}F -fluorodeoxyglucose (FDG) could be utilized for quantifying neuronal activity in the human brain (de Leon et al. 2001; Bouter et al. 2025). Therefore, exploring the relationship between SES and this fundamental aspect of brain physiology in a middle-aged cohort could provide critical insights into how SES is biologically embedded. We employed Bayesian hierarchical modelling to estimate the effects of family income and education level on brain glucose metabolism and hypothesized that family income and education level are positively associated with brain glucose metabolism.

2 | Materials and Methods

2.1 | Subjects

We retrospectively analysed data from 473 healthy males who underwent a health check-up programme at Samsung Changwon Hospital Health Promotion Center in 2013. After

excluding subjects with neuropsychiatric disorders ($n=5$) or malignancies ($n=3$), those with missing data on family income or education level ($n=232$), 233 subjects were included in this study. Subjects with missing SES data ($n=232$) were also included to compare with those with available SES data ($n=233$). The health check-up programme included (1) brain ^{18}F -FDG PET, (2) anthropometric measurements, (3) a survey of family income and education level and (4) measures of stress, anxiety and depression. Subjects in this study were included in a previous study of the effect of ageing on brain glucose metabolism (Pak et al. 2023). The study protocol was approved by the institutional review board, and informed consent from the participants was waived due to the retrospective study design.

2.2 | Brain ^{18}F -FDG PET and Image Analysis

Subjects were asked to avoid strenuous exercise for 24 h and fast for at least 6 h before the PET study. PET/CT was performed 60 min after injection of ^{18}F -FDG (3.7 MBq/kg) with the Discovery 710 PET/CT scanner (GE Healthcare, Waukesha, WI, USA). Continuous spiral CT was obtained with a tube voltage of 120 kVp and a tube current of 30–180 mA. The PET scan was obtained in three-dimensional mode with a full width at half maximum of 5.6 mm and reconstructed using an ordered-subset expectation maximization algorithm. PET scans were spatially normalized to MNI space using PET templates from SPM5 (University College of London, London, UK) with pmod version 3.6 (PMOD Technologies LLC, Zurich, Switzerland). The Automated Anatomical Labeling 2 (AAL2) atlas (Rolls et al. 2015) was used to define regions of interest (ROIs). The mean uptake of each ROI was scaled to the mean of global cortical uptake of each individual and defined as the standardized uptake value ratio (SUVR). For a full-volume analysis, the statistical threshold was set at a cluster level and corrected with a false discovery rate with $p < 0.05$ in a regression model (correction with age) after smoothing the SUVR images with a Gaussian kernel of FWHM 8 mm (Statistical Parametric Mapping 12, Wellcome Centre for Human Neuroimaging, UCL, London, UK).

2.3 | Family Income and Education Level

Self-reported family income for the last 12 months (Korean won, KRW) was converted to US dollars (USD) with an exchange rate of 1388 KRW. Education level was defined as the highest level of education completed by each subject: completion of (1) elementary school (6 years), (2) middle school (9 years), (3) high school (12 years), (4) 2-year college (14 years), (5) 4-year college (16 years) and (6) graduate school (18 years).

2.4 | Measures of Stress, Anxiety and Depression

Stress measures for the Korean National Health and Nutrition Examination Survey (KNHANES) consist of 9 self-reported items with higher scores indicating more stress ranging from 9 to 45 (Kweon et al. 2014). The Beck Anxiety Inventory is a 21 self-report measure, with higher scores indicating more anxiety ranging from 0 to 63 (Beck et al. 1988). The Centre for Epidemiologic Studies Depression Scale (CES-D) consists of 20

self-reported items with higher scores indicating more depression, ranging from 0 to 60 (Siddaway et al. 2017).

2.5 | Neurosynth Image Decoder

To test the association of family income dependent brain glucose metabolism with Neurosynth terms, we used Neurosynth Image Decoder (<https://neurosynth.org/decode>). The *t* value map of the association between brain glucose metabolism, and family income was uploaded on Neurosynth Image Decoder. The four meta-analytic uniformity maps with the highest correlation coefficients were selected to reflect the cognitive and psychological functions most strongly linked to the identified regions.

2.6 | Statistical Analysis

Normality was assessed using the Shapiro–Wilk test. Spearman correlation was used to determine the association of family income with education level, and measures of stress, anxiety and depression. We standardized the continuous variables of age, education level, family income and log-transformed regional SUVR. Mediation analysis was performed, where each potential mediator (stress, anxiety and depression) was tested separately in each model with family income or education level as a predictor and regional SUVR as the outcome. The effects of family income and education level on regional SUVR were investigated using Bayesian hierarchical modelling with brms (Bürkner 2017, 2018, 2021) that applies the Markov-Chain Monte Carlo sampling tools of RStan (Stan Development Team 2022). We set up a model for family income and education level with regional SUVR as a dependent variable and family income and education level as predictors adjusting for age. We specified custom weakly informative priors to improve model regularization: Fixed effects regression coefficients (family income, education level and age) were assigned normal priors centred at zero with

standard deviation 1 (normal(0,1)); random intercept standard deviations for subjects and regions of interest were also assigned normal(0,1) priors. These fixed effects (family income, education level and age) were calculated individually and subject and ROI were added as random intercepts to allow SUVR to vary between subjects and ROIs. Bayesian models were estimated using four Markov chains, each of which had 4000 iterations including 1000 warm-ups, thus totalling 12,000 post-warmup samples. The sampling parameters were slightly modified to facilitate convergence (max treedepth = 20). Total, direct, and indirect effects were calculated. Statistical analysis was carried out in R Statistical Software ver 4.4.1 (The R Foundation for Statistical Computing).

3 | Results

3.1 | Subjects' Characteristics

A total of 233 healthy males (mean age: 42.6 ± 3.5 years) were included in this study. The distribution of regional SUVR is shown in Figure 1. The mean family income was $61,319 \pm 17,978$ USD; 32 had less than 50,000 USD; 193 between 50,000 USD and 100,000 USD; 8 more than 100,000 USD. The mean education level was 13.6 ± 2.1 years (range 6–18 years) with 1 participant completing elementary school; 133 high school; 28 two-year college; 58 four-year college; 13 graduate school. Subjects with missing SES data ($n = 232$) were compared with those with available SES data ($n = 233$) on age, height, weight, body mass index, waist–hip ratio, measures of stress, anxiety and depression. No statistically significant differences were observed between the two groups (age, $p = 0.2022$; height, $p = 0.2658$; weight, $p = 0.5439$; body mass index, $p = 0.9297$; waist–hip ratio, $p = 0.8426$; measures of stress, $p = 0.9013$; anxiety, $p = 0.1140$; depression, $p = 0.1773$). The subjects' characteristics are summarized in Table 1. Family income was positively correlated with education level

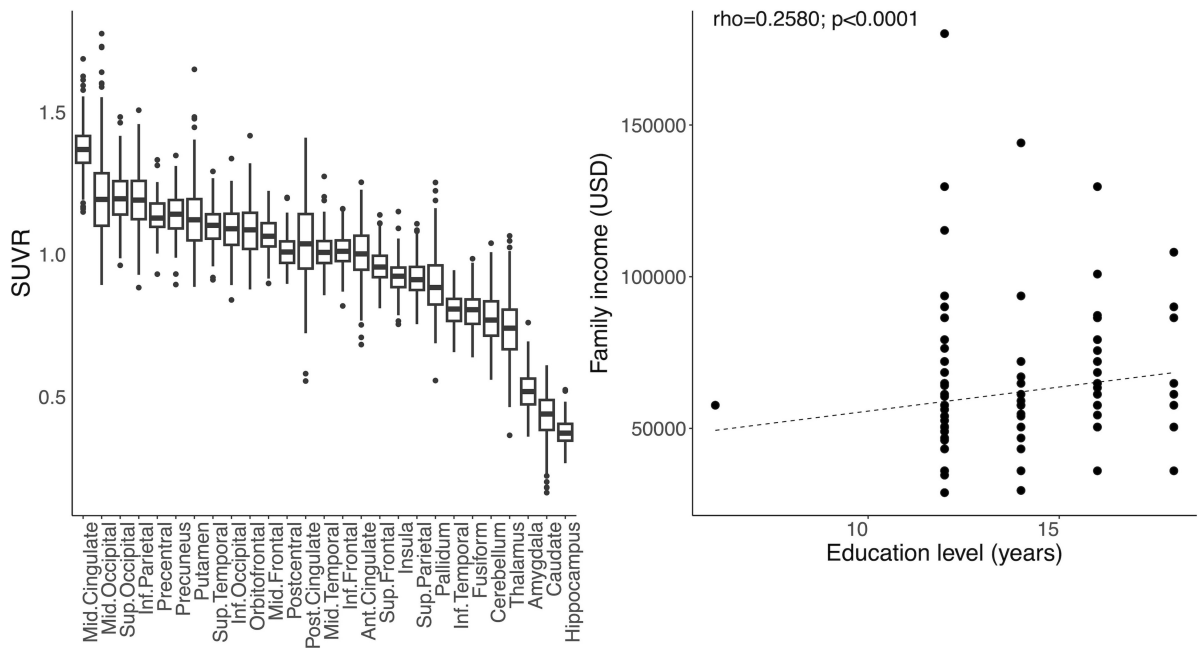


FIGURE 1 | The distribution of regional SUVR and the association between family income (USD) and education level (years).

TABLE 1 | Subjects' characteristics.

	Subjects with SES data (<i>n</i> = 233)	Subjects with missing SES data (<i>n</i> = 232)	<i>p</i>
Age (years)	42.6 ± 3.5	43.0 ± 3.7	0.2022
Height (m ²)	1.7 ± 0.1	1.7 ± 0.1	0.2658
Weight (kg)	73.2 ± 9.9	72.7 ± 9.7	0.5439
Body mass index (kg/ m ²)	24.6 ± 2.9	24.6 ± 2.8	0.9297
Waist–hip ratio	0.97 ± 0.03	0.89 ± 0.03	0.8426
Measures			
Stress	16.1 ± 6.2	16.1 ± 6.8	0.9013
Anxiety	4.9 ± 6.0	6.0 ± 7.2	0.1140
Depression	7.6 ± 5.9	6.8 ± 6.0	0.1773
Family income (USD)	61,319 ± 17,978		
Education level (years)	13.6 ± 2.1		

($\rho = 0.2580$; $p < 0.0001$), whereas the measures of stress ($\rho = 0.0084$; $p = 0.8979$), anxiety ($\rho = -0.1224$; $p = 0.0620$) and depression ($\rho = -0.0338$; $p = 0.6070$) did not show a significant association with family income (Figure 1).

3.2 | Mediation of the Association Between Family Income, Education Level and Brain Glucose Metabolism

The direct effect of family income on brain glucose metabolism was significant in the amygdala, hippocampus, pallidum and putamen ($p < 0.05$); however, that of education level was not significant in any regional brain glucose metabolism. There was no significant indirect effect of family income or education level via stress, anxiety or depression on regional brain glucose metabolism.

3.3 | Family Income, Education Level and Brain Glucose Metabolism

In a Bayesian model, family income was associated with brain glucose metabolism of fusiform (95% credible interval -0.0008 to 0.0063), putamen (95% credible interval -0.0007 to 0.0074), thalamus (95% credible interval -0.0012 to 0.0063), anterior cingulate (95% credible interval -0.0004 to 0.0069), pallidum (95% credible interval 0.0007 – 0.0088), amygdala (95% credible interval 0.0028 – 0.0119), hippocampus (95% credible interval 0.0024 – 0.0117) and caudate (95% credible interval

0.0033 – 0.0142) showing some of their 95% posterior intervals overlapping with zero. However, educational level was not associated with regional brain glucose metabolism (Figure 2). Full-volume analysis revealed the consistent finding that shows the positive association of family income with brain glucose metabolism of caudate, putamen, hippocampus, amygdala, and anterior cingulate (Figure 3).

3.4 | Localization of Family Income-Dependent Brain Glucose Metabolism and Neurosynth Terms

Meta-analytic uniformity maps of reward (correlation coefficient 0.251), monetary (correlation coefficient 0.204), incentive (correlation coefficient 0.198) and motivation (correlation coefficient 0.182) showed the most similarity with family income-dependent brain glucose metabolism. The spatial correlation between family income-dependent brain glucose metabolism and four meta-analytic uniformity maps showed the significant similarity (Figure 4).

4 | Discussion

Our main finding is that family income is positively associated with brain glucose metabolism in the caudate, putamen, anterior cingulate, hippocampus and amygdala, whereas education level does not show any significant association with brain glucose metabolism in a middle-aged male cohort. Because these findings are cross-sectional and unadjusted for health and lifestyle covariates, they should be interpreted as correlational rather than causal. Previous studies have focused on either early development or late adulthood, when the brain is most vulnerable, which are characterized by notable changes in brain structure and function (Hedman et al. 2012), leaving the effects of SES during midlife less well understood. Unlike early life, or older age, midlife offers a relatively stable phase in which to investigate how long-term exposures to SES may be associated with brain metabolism. This life stage often reflects the cumulative effects of environmental, occupational and psychosocial factors that have persisted over decades. Furthermore, emerging research suggests that changes in brain function and structure during midlife can be predictive of later-life cognitive decline and neurodegenerative disease (Singh-Manoux et al. 2012). Studying SES in this age group therefore holds promise for early identification of at-risk individuals and for informing socioeconomic and public health policies aimed at promoting brain health across the lifespan. Therefore, our finding contributes to filling this gap by providing evidence that family income, but not education level, is associated with regional brain glucose metabolism in healthy middle-aged males.

Our study demonstrates a positive association between family income, and glucose metabolism in brain regions integral to reward processing, motivation and emotional regulation, specifically the caudate, putamen, anterior cingulate, hippocampus and amygdala. These findings suggest that current socioeconomic resources may influence neural activity in circuits critical for adaptive behaviour and stress resilience. Several mechanisms may underlie these associations. First, higher family income likely buffers individuals from chronic stressors and enables

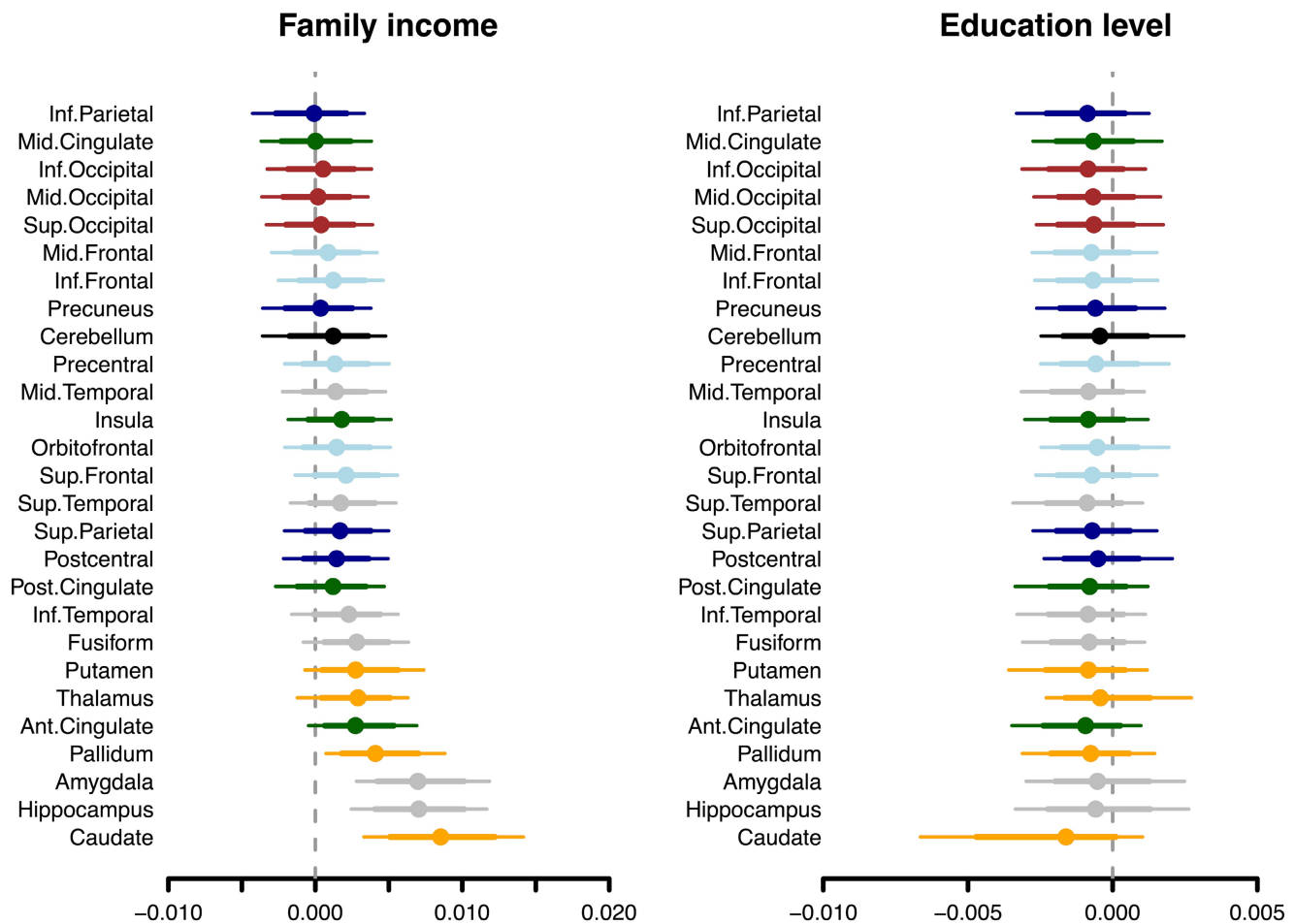


FIGURE 2 | Posterior intervals of the regression coefficients for family income and education level predicting brain glucose metabolism. The thick lines represent the 80% posterior intervals, the thin lines represent the 95% posterior intervals, and the circles represent posterior median.

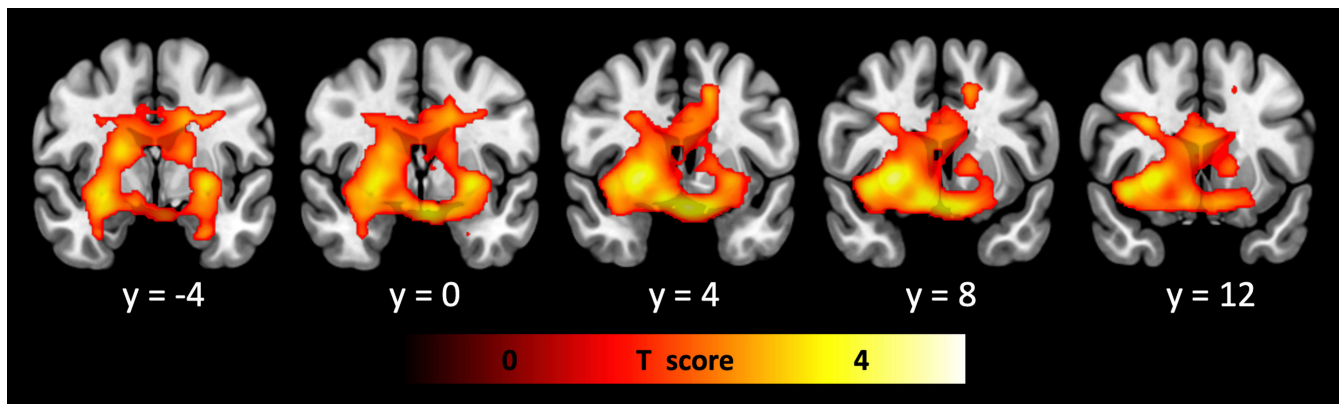


FIGURE 3 | Full volume analysis showing the positive association of family income with brain glucose metabolism.

greater access to rewarding experiences, which can enhance dopaminergic signalling and neuronal activity in reward-related brain regions. This is consistent with animal studies showing that social dominance increases DR availability (Morgan et al. 2002) and human studies indicating that higher SES is linked to greater reward sensitivity and striatal activation (Gianaros et al. 2011; Decker et al. 2024). Second, chronic financial hardship is a potent stressor that can activate the hypothalamic–pituitary–adrenal axis, leading to neurobiological changes including reduced

metabolic activity and atrophy in limbic structures such as the hippocampus and amygdala (Butterworth et al. 2012). Our findings suggest that higher family income may protect against these adverse effects, supporting greater neuronal activity in these regions. By demonstrating that family income is associated with functional as well as structural brain differences, our study extends the literature on SES and brain health, highlighting the importance of current economic resources for maintaining optimal brain function in midlife.

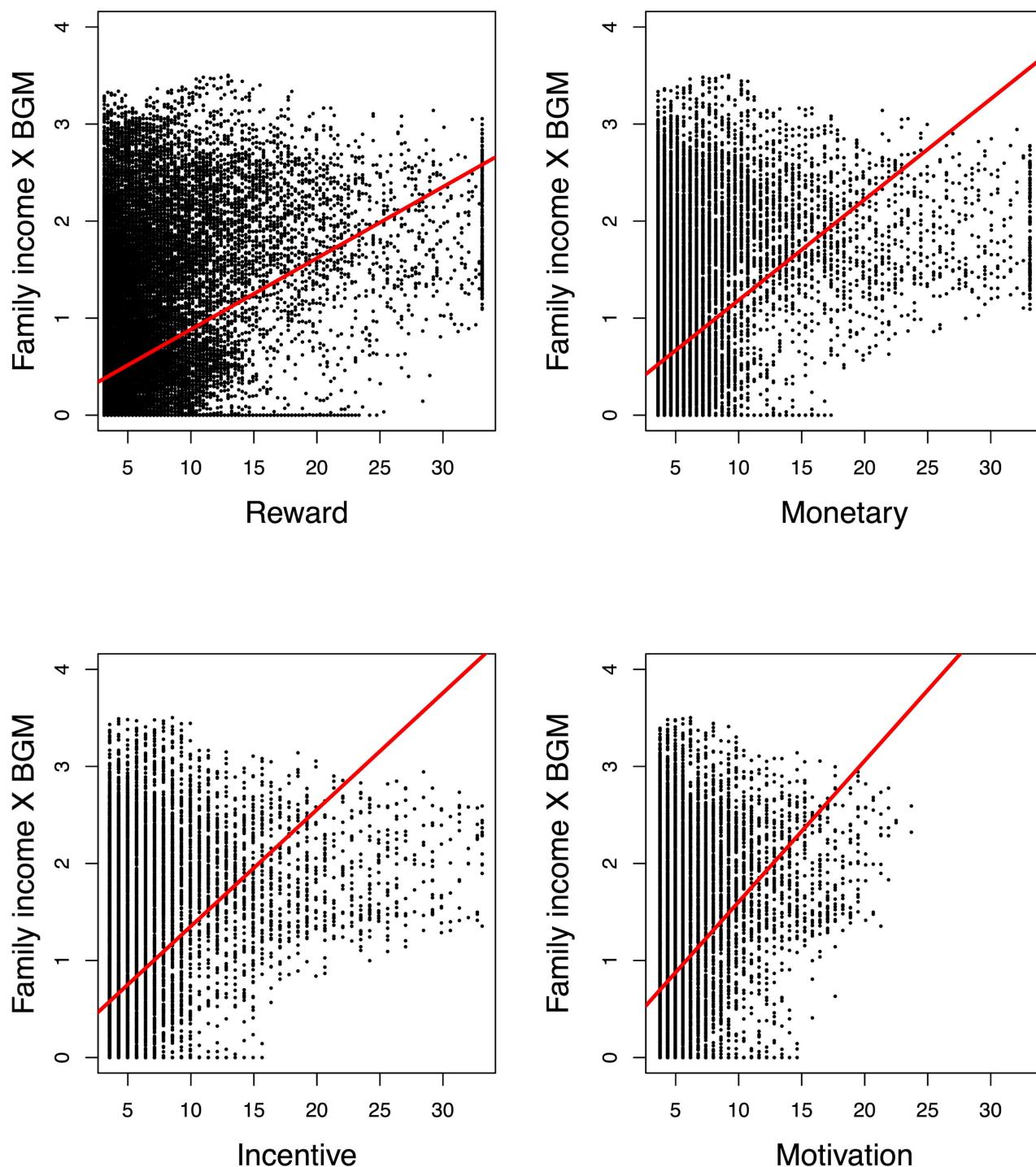


FIGURE 4 | Spatial correlation between family income-dependent brain glucose metabolism and meta-analytic blood oxygenation level-dependent functional MRI activation patterns for four terms with the most similarity retrieved from the Neurosynth Image Decoder.

Although education is often considered a key component of SES and is central to the cognitive reserve hypothesis, we did not observe significant associations between education level and brain glucose metabolism in our cohort. This finding diverges from some prior studies reporting positive associations between education and brain structure or metabolism, particularly in older adults (Arenaza-Urquijo et al. 2013; Lotze et al. 2020). Several factors may explain this discrepancy. The cognitive reserve hypothesis posits that higher education provides a buffer

against age-related or pathological brain changes, often manifesting as preserved cognitive function or neural efficiency in the face of neurodegeneration. However, most evidence for this effect comes from studies of older adults, where age-related decline is more pronounced. Our participants, with a mean age of 42.6 years, may be too young for the protective effects of education to become apparent at the level of brain metabolism. Additionally, the relatively homogeneous and high educational attainment in our sample may have limited our ability to detect

such associations. Family income, which can fluctuate and more accurately reflects current environmental exposures and stressors, may thus be a more sensitive indicator of SES-related effects on brain function in this age group.

This study has several limitations that should be considered when interpreting the findings. First, our sample consisted exclusively of males, a consequence of the health check-up programme's design at the time of data collection. Although this homogeneity reduces sex-related variability, it limits the generalizability of our results, particularly to females, who may exhibit different patterns of brain metabolism and socioeconomic influences. Prior neuroimaging research has reported sex-specific associations between family SES and brain microstructural properties with diffusion tensor imaging (Takeuchi et al. 2018). The sex-specific associations of family SES with neural and/or cognitive mechanisms particularly in neural tissues in brain areas that play key roles in basic information processing and higher-order cognitive processes in a way females with greater family SES level show imaging outcome measures that have been associated with more neural tissues and males showed the opposite (Takeuchi et al. 2018). Given these findings, our results based solely on males may not fully capture the broader spectrum of SES effects on brain glucose metabolism. Future research including both sexes is necessary to determine whether our findings extend to the broader population. Second, brain magnetic resonance imaging (MRI) was not available due to the retrospective nature of the study. The absence of MRI-based co-registration and partial volume correction for PET scans may have introduced measurement bias, potentially affecting the accuracy of regional glucose metabolism estimates. In particular, small subcortical and grey matter structures such as the caudate, putamen, hippocampus and amygdala are especially susceptible to partial volume effects due to their proximity to cerebrospinal fluid and white matter (Salminen et al. 2016). This could have resulted in an underestimation of absolute SUVs and may have attenuated or blurred true regional differences. Conversely, without precise grey-white delineation, spill-in from neighbouring regions could have led to overestimation of uptake in small structures. This limitation underscores the need for multimodal imaging in future studies to enhance anatomical precision and apply partial volume correction to improve the validity of regional metabolic quantification. Third, our assessment of SES was based on self-reported family income over the past 12 months. Although this measure captures current financial status, it may not reflect long-term or cumulative SES and is susceptible to reporting bias. Alternative indices, such as income-to-needs ratio or equivalent income, were not available, which may limit the comparability of our results to studies using different SES metrics. Additionally, nearly half (49%) of the initial cohort was excluded due to missing SES data, raising the possibility of selection bias. Individuals who completed the optional SES survey may differ systematically from those who did not, potentially influencing the observed associations. However, when we compared participants with ($n = 233$) and without ($n = 232$) SES data, no significant differences were found in age, height, weight, body mass index, waist-hip ratio, measures of stress, anxiety and depression (all $p > 0.05$), suggesting that the analytic sample is broadly representative of the original cohort. Nonetheless, the absence of SES information

for a substantial portion of the cohort still limits the generalizability of our findings and the possibility of residual selection bias cannot be entirely excluded. Another important limitation is the lack of adjustment for health and lifestyle covariates such as body mass index, physical activity and sleep patterns, all of which are known to influence brain glucose metabolism and are likely correlated with SES. Because these variables were not available in our dataset, we cannot exclude the possibility that the observed associations between family income and regional brain glucose metabolism are partly explained by these unmeasured confounders. As such, our findings should be interpreted as associations rather than evidence of a causal effect of income on brain metabolism. Lastly, the cross-sectional and observational design of our study precludes conclusions about the directionality of the relationship between family income and brain glucose metabolism. It remains unclear whether higher income leads to neurobiological changes, or whether pre-existing differences in brain function influence socioeconomic attainment. Longitudinal and interventional studies are needed to disentangle these possibilities.

5 | Conclusions

Family income and education level show differential associations with brain glucose metabolism in middle-aged males. Family income is associated with elevated brain glucose metabolism in regions involved in reward processing and stress regulation, suggesting a potential link between current socioeconomic resources and neural activity. However, these findings are cross-sectional and must be interpreted as associative rather than causal. Education level does not show a significant association with brain glucose metabolism.

Author Contributions

Kyoungjune Pak: conceptualization, methodology, software, investigation, writing – original draft, supervision. **Seunghyeon Shin:** conceptualization, formal analysis, validation, validation. **Hyun-Yeol Nam:** conceptualization, formal analysis, resources, validation. **Keunyoung Kim:** investigation, visualization. **Jihyun Kim:** methodology, writing – original draft. **Myung Jun Lee:** formal analysis, investigation. **Ju Won Seok:** investigation, writing – review and editing.

Ethics Statement

The study protocol was approved by the Institutional Review Board, and the informed consent from the participants was waived due to the retrospective study design.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data will be available from the corresponding author on reasonable request.

Peer Review

The peer review history for this article is available at <https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/ejn.70303>.

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