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The influence of receptor expression and clinical subtypes on baseline [18F]FDG uptake in breast cancer: systematic review and meta-analysis

Cornelis M. de Mooij[1,2,3*], Roxanne A. W. Ploumen[2,3], Patty J. Nelemans[4], Felix M. Mottaghy[1,3,5], Marjolein L. Smidt[2,3] and Thiemo J. A. van Nijnatten[1,3]

Abstract

Background To quantify the relationship between [18F]FDG uptake of the primary tumour measured by PET-imaging with immunohistochemical (IHC) expression of ER, PR, HER2, Ki-67, and clinical subtypes based on these markers in breast cancer patients.

Methods PubMed and Embase were searched for studies that compared SUV_{max} between breast cancer patients negative and positive for IHC expression of ER, PR, HER2, Ki-67, and clinical subtypes based on these markers. Two reviewers independently screened the studies and extracted the data. Standardized mean differences (SMD) and 95% confidence intervals (CIs) were estimated by using DerSimonian-Laird random-effects models. *P* values less than or equal to 5% indicated statistically significant results.

Results Fifty studies were included in the final analysis. SUV_{max} is significantly higher in ER-negative (31 studies, SMD 0.66, 0.56–0.77, $P<0.0001$), PR-negative (30 studies, SMD 0.56; 0.40–0.71, $P<0.0001$), HER2-positive (32 studies, SMD − 0.29, − 0.49 to − 0.10, $P=0.0043$) or Ki-67-positive (19 studies, SMD − 0.77; − 0.93 to − 0.61, $P<0.0001$) primary tumours compared to their counterparts. The majority of clinical subtypes were either luminal A (LA), luminal B (LB), HER2-positive or triple negative breast cancer (TNBC). LA is associated with significantly lower SUV_{max} compared to LB (11 studies, SMD − 0.49, − 0.68 to − 0.31, $P=0.0001$), HER2-positive (15 studies, SMD − 0.91, − 1.21 to − 0.61, $P<0.0001$) and TNBC (17 studies, SMD − 1.21, − 1.57 to − 0.85, $P<0.0001$); and LB showed significantly lower uptake compared to TNBC (10 studies, SMD − 0.77, − 1.05 to − 0.49, $P=0.0002$). Differences in SUV_{max} between LB and HER2-positive (9 studies, SMD − 0.32, − 0.88 to 0.24, $P=0.2244$), and HER2-positive and TNBC (17 studies, SMD − 0.29, − 0.61 to 0.02, $P=0.0667$) are not significant.

Conclusion Primary tumour SUV_{max} is significantly higher in ER-negative, PR-negative, HER2-positive and Ki-67-positive breast cancer patients. Luminal tumours have the lowest and TNBC tumours the highest SUV_{max}. HER2 overexpression has an intermediate effect.

Keywords Breast cancer, Immunohistochemistry, Clinical subtypes, [18F]FDG PET, Systematic review, Meta-analyses

*Correspondence:
Cornelis M. de Mooij
cmdemooij@hotmail.nl
Full list of author information is available at the end of the article

Background

Immunohistochemical (IHC) detection of estrogen receptor (ER), progesterone receptor (PR) and human epidermal growth factor receptor 2 (HER2) is the foundation of clinical subtyping of breast cancer since it selects targets for endocrine or HER2-targeted therapy [1–3]. In addition, gene expression profiling (GEP) studies have identified at least four intrinsic breast cancer subtypes that more accurately capture the diversity of breast cancer [4, 5]. Surrogate intrinsic subtypes have been defined which can be approximated using IHC determination of ER, PR, HER2 and Ki-67 [6–8]. To date, clinical subtyping using IHC has near exclusive use in contemporary practice.

Positron emission-tomography (PET) using [18F]-fluorodeoxyglucose ([18F]FDG) is a widely accepted imaging modality in breast cancer that is nowadays mostly used in combination with computed tomography (PET/CT) or magnetic resonance imaging (PET/MRI) for anatomic correlation. While mainly used for initial staging in patients with locally advanced or suspected recurrent breast cancer, it has also been thoroughly investigated for its ability to predict and detect response to neoadjuvant systemic therapy (NST) and to predict prognosis [9–11]. In practice, [18F]FDG uptake is predominantly expressed using maximum standardized uptake values (SUV_{max}).

Previous studies report a correlation of [18F]FDG uptake with tumour aggressiveness, with increased SUV_{max} in primary breast tumours that are ER-negative, PR-negative, HER2-positive or Ki-67-positive [12–14]. Studies investigating the difference in [18F]FDG uptake between clinical subtypes have found a similar pattern with relatively low SUV_{max} in subtypes including ER and PR, and high SUV_{max} for subtypes including HER2 or that are triple negative [15, 16]. To date, no meta-analysis has investigated or quantified the relative difference in SUV_{max} between IHC expression of ER, PR, HER2, Ki-67, and clinical subtypes based on these markers.

Therefore, the aim of the present study is to perform a systematic review and meta-analysis to investigate and quantify the association between [18F]FDG uptake expressed as SUV_{max} and IHC expression of ER, PR, HER2, Ki-67, and clinical subtypes based on these markers.

Methods

The full description of the methods can be obtained in Additional file 1 (Tables S1–S2). To be eligible for the meta-analysis, a study had to fulfill the following inclusion criteria: patients with invasive breast cancer, [18F]FDG uptake expressed as SUV_{max} and measured on the primary tumour before any therapy, comparison of [18F]FDG uptake between patients negative and positive for IHC expression of ER, PR, HER2, or Ki-67, and between clinical subtypes based on the IHC expression of these markers. Data on the number of patients, mean and standard deviation (SD) of SUV_{max} of patients negative and positive for IHC expression of ER, PR, HER2, Ki-67, and clinical subtypes based on these markers, was extracted. Study quality was assessed by using the Quality Assessment of Diagnostic Accuracy Studies (QUADAS)-2 tool. For the meta-analysis, the primary summary statistic was the standardized mean difference (SMD) with 95% confidence intervals (CIs) using Hedges' g correction for small study samples. The primary analyses were based on studies which presented mean [18F]FDG uptake with SD. Sensitivity analyses also included studies which presented median [18F]FDG uptake with (interquartile) range which were transformed to mean and SD. Lastly, Egger's regression test was used to identify small-study effects.

Results

Study characteristics and QUADAS-2

Figure 1 shows the search pattern and selection of articles at each step. Of the 74 included studies the means and SDs were provided in 50 [12, 14, 16–63]. In the remaining 24 studies the means and SDs were transformed from the provided medians and (interquartile) ranges [13, 64–87]. An overview of the characteristics of included studies as well as the [18F]FDG PET characteristics is provided in Additional file 2 (Tables S3–S4). The number of patients, mean and SD of each individual study for negative and positive IHC expression of ER, PR, HER2, Ki-67, and of clinical subtypes based on these markers, is provided in Additional file 2 (Tables S5–S11).

Quality of included studies

Risk of bias for patient selection originated from poor reporting of in- and exclusion criteria in three studies and the use of case–control designs in another three studies. For the index test, there was an unclear risk of bias in 26 studies since it was not reported who reviewed the PET images or performed SUV_{max} measurements, and a high risk of bias in 8 studies since no harmonization of PET-data was performed while using multiple PET-devices. With regard to the reference standard, 22 studies did not provide criteria for receptor positivity or subtypes. Lastly, high risk of bias in flow and timing existed in 8 studies since not all patients were included in the final analysis without providing valid reasons. In general, applicability concerns are low, meaning that the patient selection, index test and reference standard of the included studies match the review question. Figure 2 visualizes the risk of bias and applicability concerns and

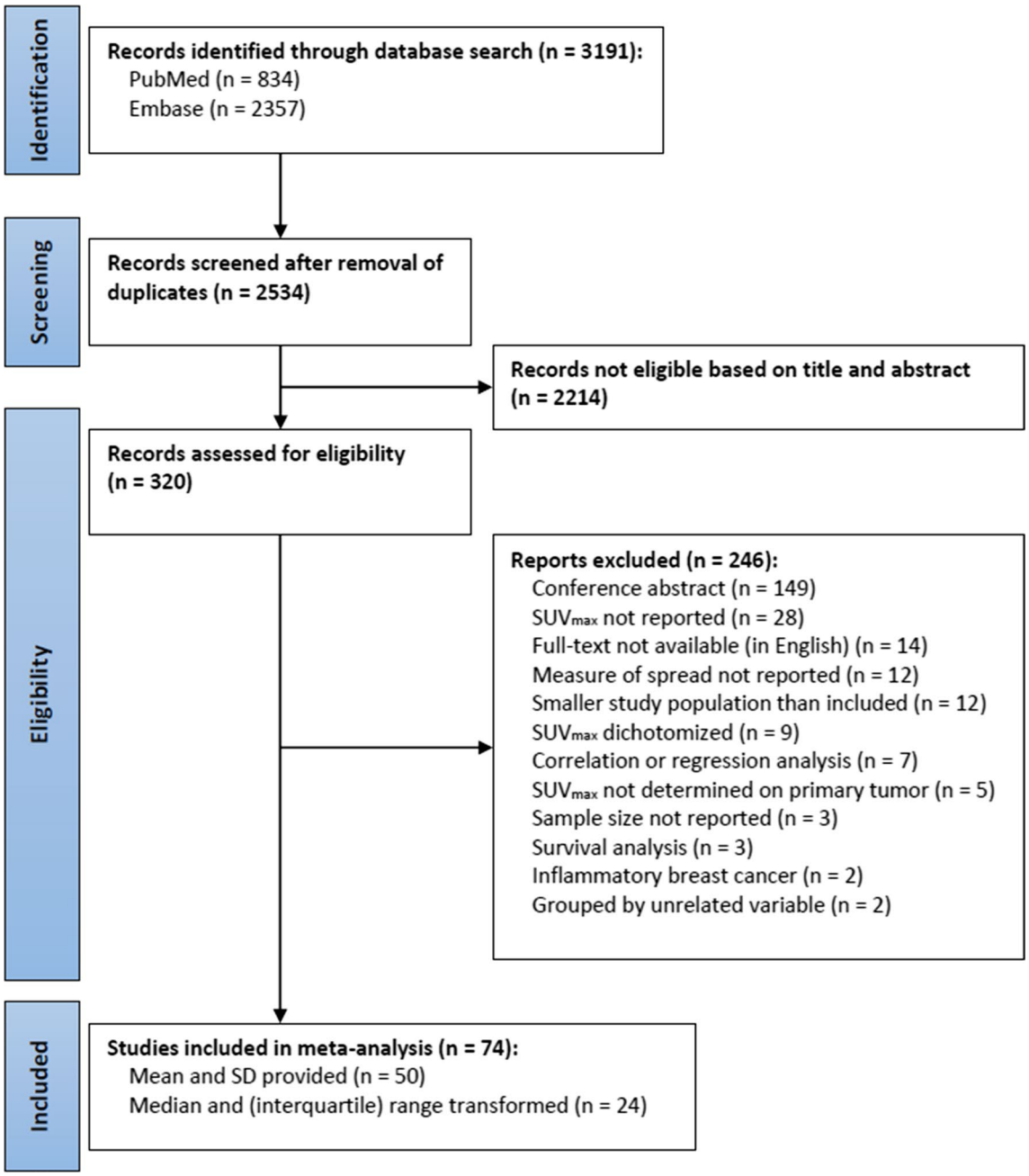

Fig. 1 PRISMA flow diagram of the study selection

additional information on methodologic quality of individual studies is provided in Additional file 2 (Table S12).

Association between [18F]FDG uptake and receptor status

Table 1 displays the estimates of the SMD with 95% CIs as measure for the difference in [18F]FDG uptake between negative versus positive IHC expression of ER, PR, HER2 and Ki-67. The primary analyses show that the SUV_{max} is significantly higher in ER-negative (SMD 0.66, $P<0.0001$), PR-negative (SMD 0.56, $P<0.0001$), HER2-positive (SMD −0.29, $P=0.0043$) or Ki-67-positive (SMD −0.77, $P<0.0001$) primary tumours compared to their counterparts.

Association between [18F]FDG uptake and surrogate intrinsic subtypes

The estimates of the SMD with 95% CIs as measure for the difference in [18F]FDG uptake between surrogate

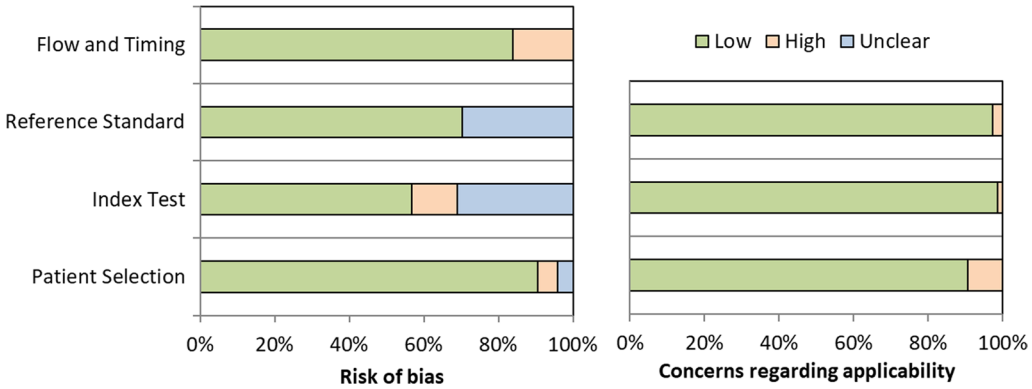

Fig. 2 Methodological quality of included studies

intrinsic subtypes based on recommendations from the St. Gallen conferences is displayed in Table 2. The primary analyses reveal that LA was associated with significantly lower SUV_{max} than LB (SMD − 0.49, $P = 0.0001$), LB HER2-negative (SMD − 0.68, $P = 0.0021$), LB HER2-positive (SMD − 0.72, $P = 0.0089$), HER2-positive (SMD − 0.91, $P < 0.0001$) and TNBC (SMD − 1.21, $P < 0.0001$); LB significantly lower than TNBC (SMD − 0.77, $P = 0.0002$); LB HER2-negative significantly lower than TNBC (SMD − 0.58, $P = 0.0177$); LB HER2-positive significantly lower than HER2-positive (SMD − 0.22, $P = 0.0457$); and TNBC significantly higher than non-TNBC (SMD 0.56, $P < 0.0001$). While the sensitivity analyses did not reveal a difference in the direction of the meta-analyses, the size and 95% CIs of the SMDs did differ significantly for the comparison of LA with LB HER2-negative ($P = 0.0213$) and of TNBC versus non-TNBC ($P = 0.0015$) when including transformed medians and (interquartile) ranges.

Association between [18F]FDG uptake and clinical subtypes according to a simplified classification

Table 3 displays the estimates of the SMD with 95% CIs as measure for the difference in [18F]FDG uptake between clinical subtypes according to a simplified classification which classified patients into three groups (i.e. ER-positive/HER2-negative, HER2-positive, and TNBC). The primary analyses reveal that SUV_{max} was significantly lower in ER-positive/HER2-negative than in HER2-positive (SMD − 0.34, $P = 0.0070$) or in TNBC (SMD − 0.89, $P = 0.0008$) and significantly lower in HER2-positive than in TNBC (SMD − 0.54, $P = 0.0193$).

Discussion

The results of this systematic review and meta-analysis indicate that there are substantial differences in [18F]FDG uptake expressed as SUV_{max} of the primary tumour between negative and positive IHC expression of ER, PR, HER2, Ki-67, and between clinical subtypes based on

Table 1 Estimates of the SMD as summary measure for the difference in [18F]FDG (SUV_{max}) uptake between negative versus positive IHC expression of ER, PR, HER2, and Ki-67

Receptor	Studies	Patients		Meta-analysis				Subgroup	Egger
	No	Negative	Positive	I^2 (%)	SMD	95% CI	*P*	*P*	*P*
Primary analyses									
ER	31	1659	3777	48.0	0.66	0.56, 0.77	< 0.0001	–	0.6530
PR	30	2043	2788	71.6	0.56	0.40, 0.71	< 0.0001	–	0.7426
HER2	32	4035	1664	80.0	− 0.29	− 0.49, − 0.10	0.0043	–	0.4726
Ki-67	19	1720	2186	57.8	− 0.77	− 0.93, − 0.61	< 0.0001	–	0.8838
Sensitivity analyses									
ER	47	2181	5256	43.1	0.67	0.59, 0.75	< 0.0001	0.7980	0.7934
PR	46	2764	4171	66.4	0.54	0.42, 0.65	< 0.0001	0.6328	0.8925
HER2	49	5602	2221	74.5	− 0.30	− 0.43, − 0.16	< 0.0001	0.9322	0.6184
Ki-67	28	2187	3028	51.0	− 0.75	− 0.87, − 0.64	< 0.0001	0.5364	0.7299

Data derived from the primary and sensitivity analyses are presented

CI, confidence interval; ER, estrogen receptor; HER2, human epidermal growth factor 2 receptor; PR, progesterone receptor; SMD, standardized mean difference

Table 2 Estimates of the SMD as summary measure for the difference in [18F]FDG (SUV_{max}) uptake between St. Gallen surrogate intrisic subtypes

Comparisons	Studies	Patients		Meta-analysis				Subgroup	Egger
	No			I^2 (%)	SMD	95% CI	*P*	*P*	*P*
Primary analyses									
LB versus									
LB	11	1022	487	29.8	−0.49	−0.68, −0.31	0.0001	–	0.8191
LBHER2−	6	234	373	32.5	−0.68	−0.97, −0.38	0.0021	–	0.4378
LBHER2+	6	234	142	54.2	−0.72	−1.17, −0.28	0.0089	–	0.2371
HER2+	15	1024	290	56.4	−0.91	−1.21, −0.61	< 0.0001	–	0.6148
TNBC	17	1054	440	63.8	−1.21	−1.57, −0.85	< 0.0001	–	0.7310
LB versus									
HER2+	9	369	185	61.2	−0.32	−0.88, 0.24	0.2244	–	0.8729
TNBC	10	405	279	36.0	−0.77	−1.05, −0.49	0.0002	–	0.5091
LBHER2− versus									
LBHER2+	6	373	142	66.0	−0.02	−0.52, 0.48	0.9316	–	0.1000
HER2+	6	373	105	51.7	−0.33	−0.81, 0.14	0.1305	–	0.4260
TNBC	6	373	157	49.5	−0.58	−1.02, −0.15	0.0177	–	0.7121
LBHER2+ versus									
HER2+	7	189	129	0.0	−0.22	−0.43, −0.01	0.0457	–	0.3950
TNBC	8	220	198	60.7	−0.45	−0.98, 0.08	0.0864	–	0.2661
HER2+ versus									
TNBC	17	326	492	58.1	−0.29	−0.61, 0.02	0.0667	–	0.6702
TNBC versus									
Non-TNBC	13	283	1157	0.0	0.56	0.41, 0.70	< 0.0001	–	0.1236
Sensitivity analyses									
LA versus									
LB	16	1361	1103	58.1	−0.46	−0.64, −0.28	< 0.0001	0.6555	0.4305
LBHER2−	7	309	522	54.9	−0.60	−0.90, −0.31	0.0025	0.0213	0.1428
LBHER2+	7	309	176	46.4	−0.71	−1.07, −0.36	0.0026	0.7466	0.3720
HER2+	21	1438	706	59.0	−0.85	−1.08, −0.62	< 00,001	0.4906	0.6625
TNBC	24	1499	865	76.5	−1.18	−1.48, −0.88	< 0.0001	0.7134	0.6259
LB versus									
HER2-pure	14	985	579	58.1	−0.37	−0.67, −0.08	0.0170	0.5380	0.3568
TNBC	15	1021	614	49.4	−0.75	−0.95, −0.55	< 0.0001	0.7621	0.2725
LBHER2− versus									
LBHER2+	7	522	176	65.3	−0.09	−0.52, 0.33	0.6078	0.1151	0.0428
HER2+	7	522	139	42.4	−0.37	−0.73, −0.01	0.0454	0.6619	0.3687
TNBC	7	522	233	45.1	−0.53	−0.86, −0.21	0.0073	0.3633	0.4680
LBHER2+ versus									
HER2+	8	223	151	0.0	−0.17	−0.37, 0.02	0.0745	0.3306	0.3426
TNBC	9	254	274	62.3	−0.37	−0.84, 0.10	0.1103	0.0792	0.1436
HER2+ versus									
TNBC	24	754	916	45.2	−0.25	−0.45, −0.06	0.0130	0.9067	0.3980
TNBC versus									
Non-TNBC	19	379	1516	40.6	0.73	0.54, 0.90	< 0.0001	0.0015	0.0526

Data derived from the primary and sensitivity analyses are presented

CI, confidence interval; HER2, human epidermal growth factor 2 receptor; LA, luminal A; LB, luminal B; SMD, standardized mean difference; TNBC, triple negative breast cancer

Table 3 Estimates of the SMD as summary measure for the difference in [18F]FDG (SUV_{max}) uptake between clinical subtypes according to a simplified classification

Comparisons	Studies	Patients		Meta-analysis				Subgroup	Egger
	No			I^2 (%)	SMD	95% CI	*P*	*P*	
Primary analyses									
ER+/HER2− versus HER2+	5	755	302	0.0	−0.34	−0.53, −0.16	0.0070	–	0.2633
ER+/HER2− versus TNBC	6	814	309	56.1	−0.89	−1.20, −0.58	0.0008	–	0.0247
HER2+ versus TNBC	5	302	291	64.7	−0.54	−0.93, −0.14	0.0193	–	0.3140
Sensitivity analyses									
ER+/HER2− versus HER2+	8	1153	416	30.9	−0.38	−0.56, −0.20	0.0016	0.3985	0.7816
ER+/HER2− versus TNBC	9	1212	424	43.0	−0.91	−1.10, −0.73	< 0.0001	0.3252	0.0246
HER2+ versus TNBC	8	416	406	22.9	−0.50	−0.76, −0.24	0.0025	0.6884	0.5186

Data derived from the primary and sensitivity analyses are presented

CI, confidence interval; ER, estrogen receptor; HER2, human epidermal growth factor receptor 2; SMD, standardized mean difference; TNBC, triple negative breast cancer

these markers. The pooled SMD estimated significantly increased SUV_{max} in tumours that are ER-negative, PR-negative, HER2-positive and Ki-67-positive. Clinical subtypes based on these markers follow the same pattern with lower SUV_{max} in luminal subtypes including ER and PR, and higher uptake in TNBC. HER2 overexpression and associated subtypes have an intermediate effect, with significantly higher uptake compared to LA and LB HER2-positive, similar uptake compared to LB and LB HER2-negative, and insignificantly lower uptake compared to TNBC.

The effect of IHC expression of each separate marker (i.e. ER, PR, HER2 and Ki-67) on [18F]FDG uptake can partially be explained by both the interrelations as well as the underlying differences in confounding clinicopathologic factors. Proliferation marker Ki-67, having the single largest effect on [18F]FDG uptake in our meta-analysis, is closely related to histological or nuclear grading and proliferative, poorly differentiated tumours are more common in ER-negative, PR-negative and HER2-positive tumours [88, 89]. In addition, tumour size has an independent effect on [18F]FDG uptake and ER-negative, PR-negative, HER2-positive, and Ki-67-positive tumours are associated with larger sizes [14, 88, 90]. This difference is further increased by an underestimation of [18F]FDG uptake in smaller tumours due to partial volume effects [91]. Lastly, invasive lobular carcinoma is associated with lower [18F]FDG uptake and is especially common in ER-positive, PR-positive and Ki-67-negative tumours [14, 92].

Clinical subtyping provides a more sophisticated classification of breast cancer compared to the separate evaluation of IHC markers. Decreased [18F]FDG uptake in luminal tumours can be attributed to ER and PR expression, with an increase in avidity in case of HER2-positivity as displayed by the increase in [18F]FDG uptake in LB and HER2-positive subtypes. Analogous to separate markers, [18F]FDG uptake closely mimicks the degree of proliferation and differentiation with a gradual increase in both [18F]FDG uptake as well as Ki-67 labeling index and poorly differentiated tumours from LA, LB, HER2-positive to TNBC [93, 94]. Paradoxically, HER2-positivity increases [18F]FDG uptake while TNBC is associated with the highest [18F]FDG uptake of all clinical subtypes. Moreover, increased [18F]FDG uptake can be attributed to larger tumours in luminal and HER2-positive subtypes, but not in TNBC due to contradictory reports on its relative tumour size compared to other subtypes [93, 94]. This suggests underlying differences in [18F]FDG uptake mechanisms between clinical subtypes beyond receptor status, tumour size, proliferation and differentiation [95].

Distinct differences in [18F]FDG uptake between clinical subtypes could influence diagnostic, predictive or prognostic performance, especially when using cutoff values to predict outcome. To illustrate, applying the same cutoff value to different clinical subtypes to predict presence of axillary lymph node metastasis (ALNM) can lead to an underestimation of performance in TNBC since this subtype is associated with increased [18F]FDG uptake and a decreased rate of ALNM [40, 96]. Contrarily, Groheux et al. reported differences in baseline as well as percentage decrease [18F]FDG uptake in primary tumour response to NST between clinical subtypes, suggesting improved diagnostic performance when using distinct cutoffs [15]. In general, the precise effect of clinical subtypes on performance of [18F]FDG PET is lacking and the results of our meta-analysis suggest a need for more research on this topic.

While practices and guidelines differ, [18F]FDG PET/CT is generally recommended in breast cancer patients

with a large primary tumour or with clinically node-positive disease [97]. While mainly performed to detect (distant) metastatic disease, the majority of primary tumours in breast cancer patients are [18F]FDG-avid [98]. In current clinical practice, [18F]FDG uptake is predominantly evaluated qualitatively. Considering the increasing number of studies reporting on the significant value of quantitative [18F]FDG PET, this imaging modality is not fully utilized by merely evaluating it qualitatively. Consequently, measuring [18F]FDG PET parameters such as SUV_{max} on the primary tumour could easily provide valuable predictive or prognostic information that could aid in clinical decision making in the context of personalized medicine. In addition, the application of artificial intelligence to [18F]FDG PET imaging provides a promising adjunct to further improve its diagnostic, predictive and prognostic accuracy [99].

The major limitations of this study were variability in the designs and methods of the included studies, specifically the variability in the administered dose of [18F]FDG, emission time, vendor, type of modality and cutoff values used for receptor status. This variability in design and methods (including vendor variability) is illustrated by the reported heterogeneities, hence the choice for SMD as a summary statistic. Including studies from 2007 onwards, differences in definitions with regard to receptor positivity as well as of criteria for clinical subtypes should be taken into account when interpreting the results of the meta-analyses in this study. Aware that varying definitions could influence the [18F]FDG uptake, there was deliberately chosen to incorporate these changes in the quality assessment instead of additional sensitivity analyses. Furthermore, it can be hypothesized that the changing criteria mainly relate to borderline cases that are of negligible effect on [18F]FDG uptake.

Conclusions

This systematic review and meta-analysis indicates a substantial and significant association between increased [18F]FDG expressed as SUV_{max} and ER-negativity, PR-negativity, HER2-positivity and Ki-67-positivity. Clinical subtypes based on these markers follow the same pattern with lower [18F]FDG uptake in luminal subtypes including ER and PR, and higher uptake in TNBC. HER2 overexpression and associated subtypes have an intermediate effect on [18F]FDG uptake. Clinical subtypes should be taken into account when applying and interpreting [18F]FDG PET in breast cancer.

Abbreviations

[18F]FDG	[18F]-fluorodeoxyglucose
ALNM	Axillary lymph node metastasis
CI	Confidence interval
CT	Computed tomography
ER	Estrogen receptor
GEP	Gene expression profiling
HER2	Human epidermal growth factor receptor 2
IHC	Immunohistochemical
LA	Luminal A
LB	Luminal B
MRI	Magnetic resonance imaging
NST	Neoadjuvant systemic therapy
PET	Positron emission tomography
PR	Progesterone receptor
QUADAS-2	Quality Assessment of Diagnostic Accuracy Studies 2
SD	Standard deviation
SMD	Standardized mean difference
SUV_{max}	Maximum standardized uptake value
TNBC	Triple negative breast cancer

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13550-023-00953-y.

Additional file 1. Full description of the methods with a delignation of the full-search algorithms for PubMed (Table S1) and Embase (Table S2).

Additional file 2. Overview of the characteristics of the included studies (Table S3), the [18F]FDG PET characteristics (Table S4), data extraction for the meta-analysis (Tables S5–S11) and methodologic quality of the included studies (Table S12).

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Author contributions

CM, ML and TN conceived the original idea and proposed the study concepts. CM and RP performed the systematic review and data extraction. CM and PN performed the meta-analysis. CM, RP and TN prepared the manuscript. CM, RP, PN, FM, ML and TN were responsible for the manuscript review. All authors have read the final manuscript and approved the version to be published.

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Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests relevant to the content of this article.

Author details

[1]Department of Radiology and Nuclear Medicine, Maastricht University Medical Centre+, P.O. Box 5800, 6202 AZ Maastricht, The Netherlands [2]Department of Surgery, Maastricht University Medical Centre+, Maastricht, The Netherlands [3]GROW – School for Oncology and Reproduction, Maastricht University Medical Centre+, Maastricht, The Netherlands [4]Department of Epidemiology, Maastricht University Medical Centre+, Maastricht, The Netherlands [5]Department of Nuclear Medicine, University Hospital RWTH Aachen University, Aachen, Germany.

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