

PERSPECTIVE ARTICLE

# Positron emission tomography/computed tomography scans: Dormant cancer activation, radiation effects, and thermography

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## Abstract

Positron emission tomography/computed tomography (PET/CT) is a cornerstone of oncologic imaging, but two concerns persist: whether diagnostic ionizing radiation could perturb tumor dormancy and whether cumulative exposure poses a meaningful stochastic risk. Infrared thermography (IRT) has also re-emerged as a potential radiation-free adjunct. This perspective article incorporates a narrative literature review. A structured PubMed search was performed using combinations of terms including PET/CT, tumor dormancy, low-dose radiation, DNA damage, angiogenesis, epidemiological risk, and IRT. Approximately 110 records were screened by title and abstract, and 68 full-text articles were reviewed. Studies were selected based on relevance to oncologic imaging, low-dose radiation biology, and translational applicability. No formal systematic review protocol or meta-analysis was applied because of heterogeneity in study design, populations, and outcomes. Experimental studies suggest that sub-Gray exposures may promote angiogenesis and dormant-to-active tumor transition; however, murine models indicate that PET-equivalent doses (~10 mGy) do not increase cancer incidence. PET/CT can induce measurable DNA damage markers, but substantial repair occurs within 24 h. Epidemiological data suggest that cumulative lymphoma surveillance exposure (~80–100 mSv) confers an incremental lifetime risk of ~0.5%, mainly from CT. IRT correlates with PET in brown adipose tissue assessment and may assist superficial tumor monitoring, but its diagnostic accuracy is lower than that of PET/CT or mammography. Overall, optimized PET/CT remains clinically indispensable, whereas artificial intelligence-enabled PET/CT-IRT integration may help guide scan timing and reduce cumulative radiation exposure.

**Keywords:** Positron emission tomography/computed tomography; Tumor dormancy; Ionizing radiation; Epidemiologic risk; Infrared thermography; Brown adipose tissue; Breast cancer detection; As low as reasonably achievable

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## 1. Introduction

Positron emission tomography combined with computed tomography (PET/CT) remains a cornerstone of oncologic imaging, providing unmatched sensitivity for staging, therapy monitoring, and restaging. Nonetheless, two key concerns persist: whether ionizing radiation—even at diagnostic levels—might influence tumor biology, and how best to balance diagnostic yield with cumulative exposure risk. An additional emerging theme is the integration of non-ionizing modalities, particularly infrared thermography (IRT), which may provide complementary physiologic information without radiation burden. This review provides a focused update for oncologists and imaging specialists, critically analyzing (i) the theoretical but biologically plausible link between low-dose radiation and the activation of dormant cancer cells, (ii) measurable biological and epidemiological effects of PET/CT exposure, and (iii) the translational role of IRT in oncology.

In this narrative review, literature was identified through PubMed using combinations of keywords such as PET/CT, tumor dormancy, low-dose radiation, DNA damage, and IRT. Only peer-reviewed articles in English were included, with priority given to studies published after 2007, when the International Commission on Radiological Protection recommendations were updated. Case reports, conference abstracts without full peer review, and non-human studies without translational value were excluded. The aim of this selection was to focus on evidence most relevant to clinical oncology and imaging practice.

## 2. Positron emission tomography/computed tomography and dormant cancer activation: Mechanistic pathways and quantitative considerations

The concept of tumor dormancy has long intrigued oncologists: micrometastatic foci may remain clinically silent for years, held in equilibrium by immune surveillance, angiogenic suppression, or cell-cycle arrest. The critical question is whether low-dose ionizing radiation from diagnostic imaging might disturb this equilibrium, inducing a transition from quiescence to active proliferation.

### 2.1. Angiogenic switch induced by low-dose radiation

Experimental data suggest that even sub-Gray exposures can modify endothelial cell behavior. In vitro assays show that doses of 0.5–0.8 Gy enhance migration velocity and branching morphogenesis.<sup>1</sup> From a quantitative perspective, endothelial migration rates ( $v$ ) can be approximated by:

$$v = v_0 \cdot e^{\alpha D} \quad (1)$$

where  $v_0$  is basal migration speed,  $D$  is radiation dose, and  $\alpha$  is a dose-sensitivity constant derived from empirical fits. For endothelial cells,  $\alpha$  values are estimated at 0.1–0.3 Gy<sup>−1</sup>, suggesting that even modest increases in dose accelerate angiogenesis exponentially at low doses. Such shifts could, in theory, tip dormant tumors past the “angiogenic threshold” required for growth.

This formulation assumes a simplified exponential relationship between radiation dose and endothelial migration, derived from *in vitro* observations. It does not account for tissue heterogeneity, immune modulation, or dose-rate effects, and therefore serves only as a conceptual illustration rather than a predictive clinical model.

### 2.2. Dormancy escape and population kinetics

Dormant micrometastases can be modeled as a balance between proliferation rate ( $P$ ) and apoptosis rate ( $A$ ). A steady state exists when  $P = A$ . Radiation-induced microenvironmental changes may increase  $P$  via growth factor release or decrease  $A$  via DNA repair stimulation. The transition probability from dormancy ( $P = A$ ) to active growth ( $P > A$ ) can be estimated using stochastic birth–death models:

$$P_{\text{escape}}(t) \approx 1 - e^{-(P - A)t} \quad (2)$$

When  $P = A$ , the escape probability is near zero. A small radiation-induced shift where  $P - A = 0.01 \text{ day}^{-1}$  could yield a ~30% chance of dormancy escape over a single year, underscoring how small biological perturbations may accumulate clinically.

This stochastic model assumes constant proliferation and apoptosis rates over time and does not incorporate immune surveillance or microenvironmental feedback. As such, it highlights theoretical sensitivity to small perturbations but should not be interpreted as a direct predictor of clinical dormancy escape.

### 2.3. Diagnostic versus experimental dose scales

For clarity, it is important to distinguish between absorbed dose (Gy or mGy), which reflects the energy deposited in tissue, and effective dose (mSv), which accounts for organ sensitivity and stochastic risk weighting. Experimental radiobiology studies often report absorbed doses in Gy, whereas diagnostic imaging risk is conventionally expressed in mSv. In PET/CT, fluorodeoxyglucose (FDG) contributes primarily to absorbed dose, while effective dose—and thus cancer risk estimation—is dominated by the CT component.

It is crucial, however, to distinguish experimental low-dose ionizing radiation doses from diagnostic PET exposures. While *in vitro* pro-angiogenic effects manifest at 0.5 Gy, the whole-body effective dose from FDG-PET is typically 7–10 mSv (0.007–0.01 Gy). This discrepancy—two orders of magnitude—suggests that mechanistic plausibility at higher low-dose levels does not directly translate into diagnostic PET. It is important to clarify that most experimental data on radiation-induced angiogenesis and dormancy escape were obtained at sub-Gray or higher exposures, which are not directly comparable to diagnostic PET/CT doses. Therefore, these findings demonstrate biological plausibility but should not be interpreted as evidence that clinical PET exposures can activate dormant tumors.

Supporting this, cancer-prone murine models exposed to PET-equivalent doses (~10 mGy) exhibited no increase in tumor frequency nor shortened latency.<sup>2</sup> These results imply that the dose–response curve for dormancy activation is non-linear, with a practical threshold well above diagnostic imaging ranges.

#### 2.4. Systemic effects and the “Badscopal” analogy

At therapeutic levels, radiation has been shown to paradoxically stimulate distant tumor growth, mediated by factors such as amphiregulin.<sup>3</sup> The probability of such systemic effects can be framed by diffusion–reaction equations for cytokine propagation:

$$\partial C/\partial t = D_c \nabla^2 C - \lambda C + S(x, t) \quad (3)$$

where  $C$  is cytokine concentration,  $D_c$  is diffusion constant,  $\lambda$  is degradation rate, and  $S(x, t)$  represents source production in irradiated tissue. Although diagnostic PET lacks the dose intensity to produce strong systemic cytokine gradients, the analogy highlights that radiation is not purely local in its biological consequences.

### 3. Biological effects and risk from positron emission tomography/computed tomography

#### 3.1. Immediate cellular and molecular responses

Positron emission tomography/computed tomography delivers ionizing radiation via two components: the positron-emitting radiotracer (e.g., <sup>18</sup>F-FDG) and the CT scan. Even at diagnostic doses, radiation exposure produces measurable DNA double-strand breaks (DSBs) in circulating lymphocytes, which are detectable by  $\gamma$ -H2AX foci assays.<sup>4</sup> Chromosomal aberrations have also been observed post-PET, suggesting that these doses are

biologically non-trivial.

From a kinetic standpoint, DNA damage and repair dynamics can be modeled as:

$$dN/dt = k_{\text{ind}} D - k_{\text{rep}} N \quad (4)$$

where  $N$  is the number of DSBs,  $k_{\text{ind}}$  is the induction constant proportional to dose  $D$ , and  $k_{\text{rep}}$  is the repair rate constant. For example, the time-dependent reduction of  $\gamma$ -H2AX foci reflects rapid repair kinetics of PET/CT-induced DSBs and is consistent with a high  $k_{\text{rep}}$ , with substantial resolution of damage observed within 24 h.<sup>4</sup> This rapid resolution explains why the clinical consequences of such damage remain uncertain.

#### 3.2. Cumulative exposure and epidemiological risk

While a single PET/CT conveys modest risk, repeated scans in cancer follow-up accumulate significant effective doses. In lymphoma cohorts, cumulative exposures of ~80–100 mSv over several years were associated with modeled increases in lifetime attributable cancer risk.<sup>5</sup>

The probability of radiation-induced malignancy can be estimated by the linear no-threshold model:

$$P_{\text{cancer}} = \beta \cdot D_{\text{eff}} \quad (5)$$

where  $D_{\text{eff}}$  is the effective dose and  $\beta$  is the risk coefficient, approximately  $5 \times 10^{-2}$  per Sv.<sup>6</sup> For example, a patient receiving 100 mSv cumulative dose has an estimated 0.5% incremental lifetime cancer risk. While small, this is not negligible—particularly in younger cohorts with longer life expectancy.

Clinical data in lymphoma patients underscore the epidemiological dimensions of PET/CT radiation exposure. In a cohort study, Nievelstein *et al.*<sup>7</sup> evaluated mortality risk and concluded that while PET/CT + CT imaging in malignant lymphoma introduces modest radiation-induced risk, this is largely justified by the benefits of imaging—yet the risk remains quantifiable, especially in younger or long-lived patient populations, necessitating judicious use of imaging.<sup>7</sup>

Further, in breast imaging, Omranipour *et al.*<sup>8</sup> directly compared thermography vs. mammography: thermography showed sensitivity similar to mammography (~81.6% versus ~80.5%) but significantly lower specificity (57.8% versus 73.3%), and overall lower diagnostic accuracy (69.7% versus 76.9%)—suggesting that while thermography has clinical value as adjunct or screening complement, it cannot replace mammography.<sup>8</sup>

Moreover, it is essential to emphasize that in PET/CT imaging, the majority of radiation risk derives from the CT component, not the radiotracer FDG. Studies such as that by Nievelstein *et al.*<sup>7</sup> delineate that FDG contributes only a small fraction of the effective dose; the CT acquisition (especially for diagnostic or full-body staging) often dominates the cumulative dose burden. Thus, risk mitigation strategies should prioritize CT protocol optimization, reducing CT scan length or voltage, and, where possible, use of low-dose CT, rather than focusing solely on tracer activity.

The mathematical models applied in this review, including the linear no-threshold model and stochastic birth–death kinetics, are theoretical frameworks. These models are not yet validated in large clinical cohorts, and their predictions should be interpreted with caution. This limitation should be considered when extrapolating biological plausibility to real-world oncology patients.

### 3.3. Organ-specific doses and stochastic effects

Positron emission tomography radiotracers preferentially accumulate in metabolically active tissues. For FDG, the bladder wall often receives the highest absorbed dose (~20–30 mGy per scan) due to urinary excretion.<sup>9</sup> Stochastic models predict that tissues with higher absorbed doses may exhibit proportionally greater relative risk.

Another important limitation is that organ-specific absorbed doses were not estimated in detail using the medical internal radiation dose schema. The effective dose provides a global measure of risk, but does not account for critical organ exposure such as the bladder wall, thyroid, or gonads. Future analyses should include medical internal radiation dose-based dosimetry to provide a more precise evaluation of stochastic and deterministic risks in PET/CT.

A Poisson distribution can approximate the probability of mutational “hits”:

$$P(k; \mu) = \mu^k e^{-\mu} / k! \quad (6)$$

where  $k$  is the number of DNA lesions in a given cell and  $\mu = \lambda D$ , with  $\lambda$  being the lesion yield per Gy. At diagnostic doses (0.01 Gy),  $\mu$  is small ( $<0.1$ ), meaning most cells sustain zero or one lesion—readily repairable—but repeated scans increase the cumulative probability of lesions.

### 3.4. Dose optimization and clinical implications

Modern PET/CT systems employ strategies such as time-of-flight imaging and digital detectors, enabling reduced injected activity and lower CT dose. Clinical studies demonstrate that optimized protocols can halve patient dose while maintaining diagnostic accuracy.<sup>10–11</sup>

The balance between diagnostic benefit and radiation risk can be formalized as a benefit–risk ratio ( $BRR$ ):

$$BRR = U_{\text{diagnostic}} / R_{\text{stochastic}} \quad (7)$$

where  $U_{\text{diagnostic}}$  is the utility of PET in altering patient management, and  $R_{\text{stochastic}}$  is the modeled long-term cancer risk. In PET/CT,  $BRR$  is overwhelmingly  $> 1$  in oncology, reaffirming its clinical justification when appropriately indicated.

## 4. Infrared thermography as a complementary modality

### 4.1. Physiological basis and biothermal principles

Infrared thermography detects electromagnetic radiation in the 8–14  $\mu\text{m}$  spectrum, providing a non-ionizing, surface-level surrogate of tissue perfusion and metabolism. The physiological rationale is that tumors and activated brown adipose tissue (BAT) exhibit hypervascularity and increased metabolic heat production, altering local skin surface temperature.

The relationship between metabolic heat generation ( $Q$ ) and surface temperature elevation ( $\Delta T$ ) can be modeled by the Pennes’ bioheat equation:

$$pc \cdot \partial T / \partial t = k \nabla^2 T + \omega_b c_b (T_a - T) + Q_m \quad (8)$$

where  $p$  and  $c$  are tissue density and specific heat,  $k$  is thermal conductivity,  $\omega_b$  is blood perfusion rate,  $c_b$  is blood specific heat,  $T_a$  is arterial blood temperature, and  $Q_m$  is metabolic heat production.

In tumors, increases in  $Q_m$  and  $\omega_b$  lead to localized  $\Delta T$  detectable by IRT, typically in the range of 0.5–2.0  $^{\circ}\text{C}$  compared to contralateral normal tissue.<sup>12–13</sup>

### 4.2. Correlation with positron emission tomography and brown adipose tissue studies

Several studies have validated IRT against  $^{18}\text{F}$ -FDG PET for the assessment of BAT activation. Law *et al.*<sup>14</sup> demonstrated that supraclavicular thermal hotspots measured by IRT correlated strongly with PET SUVmax ( $r \approx 0.7$ ). This correlation supports the concept that IRT is a non-ionizing proxy for BAT metabolic imaging, with potential to reduce the need for repeated PET scans in longitudinal BAT studies.

Quantitatively, if PET SUV is proportional to glucose uptake ( $U_g$ ), and IRT  $\Delta T$  reflects perfusion-related heat, the correlation can be approximated as:

$$U_g \propto \Delta T^\gamma \quad (9)$$

where  $\gamma$  is an empirically derived exponent ( $\approx 1.2$  in cold-activation BAT studies). This highlights the quasi-linear coupling between PET metabolic data and thermal signatures.

Recent systematic evidence has also highlighted the potential of IRT in the functional assessment of BAT. In a review, 12 eligible studies were analyzed, and the majority reported a positive correlation between elevations in supraclavicular skin temperature and PET-derived measures of BAT activation.<sup>15</sup> Although methodological heterogeneity precluded meta-analysis, the review emphasized that IRT represents a promising radiation-free alternative for BAT monitoring and underscored the need for methodological standardization to improve

reproducibility. This body of evidence strengthens the argument that thermography, while not a replacement for PET/CT, may offer practical value in specific scenarios where repeated, non-invasive, and radiation-free assessments are advantageous.

#### 4.3. Oncology applications: Superficial tumors and inflammation

Pilot clinical studies have explored IRT in detecting superficial breast and skin tumors. Omranipour *et al.*<sup>8</sup> reported that IRT could identify malignant lesions with a sensitivity of 81.6%, specificity of 57.8%, positive predictive value of 78.9%, and negative predictive value of 61.9% compared to histopathology. Although performance is lower than PET/CT, IRT provides real-time, radiation-free adjunctive data (Figure 1).

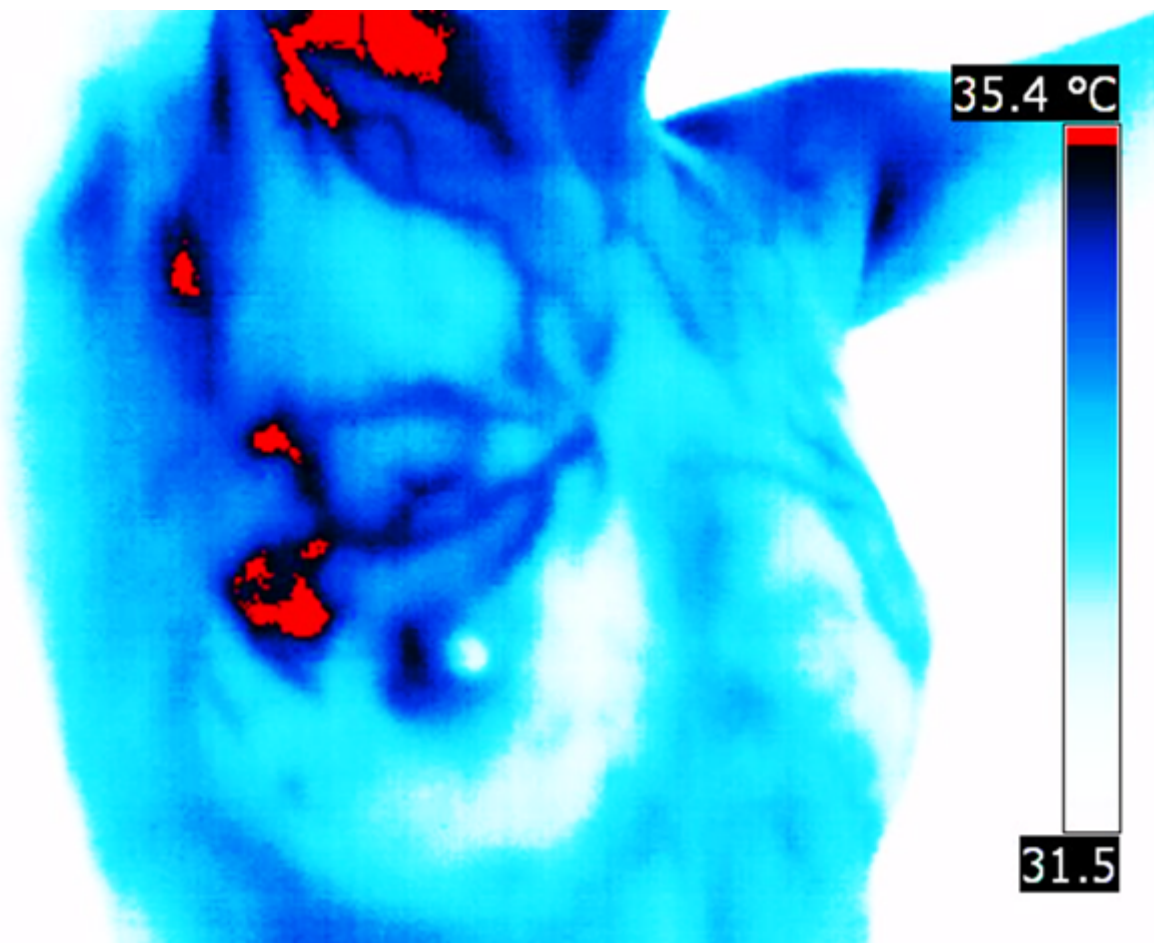


**Figure 1.** Infrared thermography of a patient with histologically confirmed invasive ductal carcinoma of the right breast, captured at the initiation of radiotherapy. The thermal map shows asymmetric hyperperfusion regions, with vascular patterns and increased metabolic activity in the affected breast compared to the contralateral side. This image illustrates the potential of infrared thermography as a complementary tool for documenting perfusion and metabolic changes in oncologic patients undergoing treatment (image from the authors).

In oncology follow-up, IRT may also serve as a functional biomarker of therapy response, as early reductions in vascularity and perfusion manifest as cooling patterns before volumetric shrinkage appears on CT or magnetic resonance imaging.<sup>16</sup>

#### 4.4. Clinical limitations and regulatory stance

Despite promise, IRT suffers from limited depth penetration, high inter-patient variability, and environmental sensitivity. The Food and Drug Administration explicitly cautions that thermography must not replace mammography or other established screening tools, underscoring its role as a complementary modality.<sup>17</sup> Although infrared thermography shows relatively low specificity compared with PET/CT and mammography, this limitation does not eliminate its potential clinical utility. Because IRT is a non-ionizing technique, it represents a safe and repeatable tool. For this reason, the scientific community should continue to explore its potential as a complementary method, especially for longitudinal follow-up where radiation burden must be minimized.

From a Bayesian diagnostic perspective, the posterior probability of malignancy given a positive IRT ( $P(M|IRT^+)$ ) is highly dependent on the pre-test probability:

$$P(M|IRT^+) = S_e \cdot P(M) / S_e \cdot P(M) + (1 - S_p)(1 - P(M)) \quad (10)$$

where  $S_e$  is sensitivity,  $S_p$  is specificity, and  $P(M)$  is baseline prevalence. In high-prevalence cohorts (e.g., oncology clinics), IRT can meaningfully increase diagnostic confidence. In screening populations with low prevalence, however, false positives dominate.

Another relevant aspect is occupational radiation exposure. PET/CT procedures involve handling radiopharmaceuticals and operating CT scanners, which result in measurable doses to healthcare staff. Studies show that nuclear medicine technologists and radiologists may accumulate non-negligible exposure over time.<sup>18</sup> In this context, IRT offers a clear advantage as it is completely radiation-free, not only for patients but also for staff, and could help reduce the frequency of PET/CT procedures in specific scenarios.

A meta-analysis or quantitative synthesis was not performed in this review because of heterogeneity in study designs, populations, imaging protocols, and outcome measures. The available literature includes animal models, cellular studies, and clinical cohorts, which cannot be combined statistically without introducing bias. For this reason, the review remains narrative, and this must be acknowledged as a limitation.

## 5. Conclusion

The cumulative evidence reviewed suggests that diagnostic PET/CT is unlikely to trigger dormant tumor activation under clinically relevant conditions. While mechanistic studies demonstrate that low-dose ionizing radiation can influence angiogenic and cellular pathways, these effects generally occur at exposure levels substantially higher than those used in diagnostic PET imaging. Mechanistic studies suggest that low-dose ionizing radiation can promote endothelial migration and angiogenic signaling, but these phenomena occur at exposures far exceeding the 7–10 mSv typical of PET scans. Animal models support that PET-equivalent doses are biologically safe, supporting the robust use of this modality in oncology.

Nevertheless, PET/CT is biologically active at the molecular level, inducing measurable DNA damage markers and chromosomal aberrations. The linear no-threshold model predicts that repeated surveillance can incrementally increase lifetime attributable cancer risk by ~0.5% in heavily imaged cohorts. Although modest, these risks highlight the ongoing need for strict adherence to as low as reasonably achievable principles and optimization of dose protocols.

Infrared thermography emerges as a radiation-free adjunct, rooted in the physics of bioheat transfer and validated against PET in BAT research. Its applications in superficial tumor detection and therapy monitoring are promising, especially when quantified mathematically through models such as the Pennes' bioheat equation. However, its limitations in depth penetration and specificity confirm that IRT remains complementary and is not a substitute for PET/CT or MRI.

The future outlook lies in hybrid imaging strategies. PET/CT continues as the gold standard for deep metabolic profiling, while IRT can provide frequent, radiation-free physiologic monitoring between scans. By integrating artificial intelligence (AI) and Bayesian diagnostic modeling, this synergy could minimize cumulative radiation exposure while enriching functional insight into tumor biology.

Positron emission tomography/computed tomography remains the gold standard for metabolic imaging and tumor staging. IRT is not suitable as a stand-alone alternative, but when used in combination with PET/CT and AI algorithms, it may provide additional value for superficial tumors such as breast cancer, thyroid nodules, and cervical or inguinal lymph nodes. Recent advances in AI-based classification and reinforcement learning applied to oncologic imaging further support this direction, as demonstrated by Muksimova *et al.*<sup>19</sup> who developed a novelty classification

model for cervical cancer using reinforcement learning approaches, reinforcing the relevance of AI in cancer imaging workflows. Recent evidence supports the role of multimodal imaging strategies in oncology.<sup>20–22</sup>

In summary, PET/CT remains indispensable for cancer staging and management, with an excellent benefit–risk ratio. IRT may evolve into a valuable adjunct, especially in precision oncology and longitudinal monitoring. Together, these approaches illustrate the need for multimodal imaging strategies that balance diagnostic accuracy, biological safety, and patient-centered care.

Artificial intelligence-driven multimodal fusion of PET/CT and IRT should currently be considered a promising investigational framework. Further prospective validation, standardization of acquisition protocols, and regulatory evaluation will be required before clinical implementation.

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## Conflict of interest

The authors declare that they have no competing interests.

## Author contributions

*Conceptualization:* All authors

*Visualization:* Marcos Leal Brioschi, Gabriel Carneiro Brioschi

*Writing—original draft:* All authors

*Writing—review & editing:* All authors

## Ethics approval and consent to participate

This article is a narrative review and did not involve direct research with human participants, animals, or cell lines. Ethical approval and consent to participate were not applicable.

## Consent for publication

Written informed consent was obtained from the patient for publication of the thermographic image presented in [Figure 1](#).

## Availability of data

Not applicable.

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