MedCrave
Step into the World of Research

Journal of Cancer Prevention & Current Research

Review article

Open Access

CrossMark

PET tracers: radiation exposure and malignancy risk

Abstract

Positron emission tomography/computed tomography exposes patients to two sources of ionizing radiation: internally administered PET radiotracers and externally delivered CT radiation. Although the radiotracer contribution can generally be estimated from administered activity, CT exposure varies substantially according to scan coverage, patient size, equipment, acquisition parameters, and whether the CT is performed for attenuation correction or as a diagnostic-quality examination. This review presents representative effective doses for commonly used PET radiotracers and estimates the associated lifetime attributable risk of malignancy for men and women exposed at age 40 using the Biological Effects of Ionizing Radiation VII model. The CT component may equal or exceed the radiotracer contribution and can substantially increase the total procedural dose. Recent biological evidence demonstrates detectable chromosomal injury following standard-dose PET/CT but not reduced-dose PET/CT, although the absence of a detectable FDG-only effect does not establish the absence of biological injury. Repeated PET/CT examinations may produce substantial cumulative exposure. Current risk models combine event-specific risks but do not provide a validated interval-dependent curve describing how examinations separated by weeks, months, or years interact biologically. Transparent patient counseling should therefore address the total PET/CT exposure, the anticipated CT protocol, the possibility of additional CT examinations, and the cumulative exposure expected from serial imaging.

Keywords: positron emission tomography; computed tomography; radiotracer; effective dose; radiation exposure; lifetime attributable risk; malignancy; repeated imaging

Volume 17 Issue 4 - 2026

Richard C Semelka,[1] Miguel Ramalho[2]
[1]Richard Semelka Consulting, PLLC, USA
[2]Department of Radiology, Hospital da Luz, Portugal

Correspondence: Richard Semelka MD, Richard Semelka Consulting, PLLC, 3901 Jones Ferry Road, Chapel Hill, NC, USA, Tel 9196028792

Received: July 27, 2026 | **Published:** August 10, 2026

Introduction

Existing PET radiotracers and novel molecularly targeted agents have generated considerable interest for imaging patients with cancer, dementia, neuroendocrine tumors, and other disorders. What is generally lacking, particularly in patient-facing information, is a transparent description of the actual radiation exposure associated with these examinations. This information is necessary so that an individual can make an informed decision regarding whether the expected diagnostic or management benefit justifies the procedural radiation risk.

The radiation exposure from a PET/CT examination has two separate components: the internally administered PET radiotracer and the CT acquisition. The radiotracer dose is relatively predictable when the administered activity is known. In contrast, the CT exposure varies tremendously according to the body region examined, scan length, patient size, scanner technology, tube current and voltage, whether dose modulation is used, and whether the CT is performed only for attenuation correction or as a diagnostic-quality examination. Protocol optimization also varies among imaging facilities, and actual CT exposures may therefore fall toward or above the higher end of published ranges.

Age 40 is used as a standardized reference age for estimating radiation-associated malignancy risk. Although radiation risks are often reported only for men or as a combined-sex approximation, the present table reports separate values for men and women. The estimates are based on the Biological Effects of Ionizing Radiation VII report and represent lifetime attributable risk of cancer incidence following exposure at age 40.[1]

It must be emphasized that many PET exams are of clinical importance to patient care, and careful risk/ benefit evaluation must be made. Part of the dialogue should include the radiation risk, albeit often small, in patient discussions. This issue will be elaborated in the Discussion (Table 1).

Table 1 PET radiotracer radiation exposure and estimated lifetime malignancy risk

PET radiotracer and representative use	PET radiation exposure (mSv)*	Representative total PET/CT exposure (mSv)†	Estimated excess malignancy risk: 40-year-old male‡	Estimated excess malignancy risk: 40-year-old female‡
^{2}F-FDG, 370 MBq — general oncology, infection/inflammation, cardiac and brain imaging	7	12.0–17.0	78–110 per 100,000; approximately 1 in 1,300 to 1 in 910	106–151 per 100,000; approximately 1 in 940 to 1 in 660
^{2}F-Piflufolastat, PYLARIFY, 370 MBq — PSMA-positive prostate cancer	4.3	9.3–14.3	60–93 per 100,000; approximately 1 in 1,660 to 1 in 1,080	82–127 per 100,000; approximately 1 in 1,210 to 1 in 790§
^{2}Ga-Gozetotide/PSMA-11, LOCAMETZ, 259 MBq — PSMA-positive prostate cancer	4.4	9.4–14.4	61–93 per 100,000; approximately 1 in 1,640 to 1 in 1,070	83–128 per 100,000; approximately 1 in 1,200 to 1 in 780§

Table 1 Continued...

^{2}F-Flotufolastat, POSLUMA, 296 MBq — PSMA-positive prostate cancer	4.1	9.1–14.1	59–91 per 100,000; approximately 1 in 1,700 to 1 in 1,090	81–125 per 100,000; approximately 1 in 1,240 to 1 in 800§
^{2}F-Fluciclovine, AXUMIN, 370 MBq — suspected recurrent prostate cancer	8	13.0–18.0	84–117 per 100,000; approximately 1 in 1,190 to 1 in 860	115–159 per 100,000; approximately 1 in 870 to 1 in 630§
^{2}F-Fluoroestradiol, CERIANNA, 222 MBq — estrogen-receptor–positive recurrent or metastatic breast cancer	4.9	9.9–14.9	64–97 per 100,000; approximately 1 in 1,560 to 1 in 1,040	88–132 per 100,000; approximately 1 in 1,140 to 1 in 760
^{2}F-Florbetapir, AMYVID, 370 MBq — cerebral amyloid imaging	7	8.0–9.5	52–62 per 100,000; approximately 1 in 1,930 to 1 in 1,620	71–84 per 100,000; approximately 1 in 1,410 to 1 in 1,190
^{2}F-Florbetaben, NEURACEQ, 300 MBq — cerebral amyloid imaging	5.8	6.8–8.3	44–54 per 100,000; approximately 1 in 2,270 to 1 in 1,860	60–74 per 100,000; approximately 1 in 1,660 to 1 in 1,360
^{2}F-Flutemetamol, VIZAMYL, 185 MBq — cerebral amyloid imaging	5.9	6.9–8.4	45–54 per 100,000; approximately 1 in 2,240 to 1 in 1,840	61–74 per 100,000; approximately 1 in 1,640 to 1 in 1,340
^{2}F-Flortaucipir, TAUVID, 370 MBq — cerebral tau imaging	8.7	9.7–11.2	63–73 per 100,000; approximately 1 in 1,590 to 1 in 1,380	86–99 per 100,000; approximately 1 in 1,160 to 1 in 1,010
^{2}Ga-DOTATATE, NETSPOT, 150 MBq — somatostatin-receptor–positive neuroendocrine tumors	3.2	8.2–13.2	53–86 per 100,000; approximately 1 in 1,880 to 1 in 1,170	73–117 per 100,000; approximately 1 in 1,380 to 1 in 860

Table footnotes

PET radiotracer exposure. The values shown are representative effective doses at the stated administered activities. Actual exposure varies with the activity administered, patient anatomy, biokinetics, hydration, frequency of bladder emptying, and the dosimetric model employed. For ^{2}F-FDG, an adult effective-dose coefficient of approximately 0.019 mSv/MBq was used.[3] Modern scanners may permit lower administered activities than the reference activities listed.

Total PET/CT exposure. For whole-body or skull-base-to-thigh oncologic PET/CT examinations, the tabulated range assumes that the CT component contributes approximately 5–10 mSv. For dedicated dementia brain PET/CT, the CT component is assumed to contribute approximately 1–2.5 mSv. The US Food and Drug Administration reports a typical adult effective dose of approximately 2 mSv for diagnostic head CT.[4]

The tabulated values should not be interpreted as an upper limit. Depending on scan length, patient size, equipment, number of phases, use of intravenous contrast, and facility-specific dose optimization:

1. A diagnostic-quality or less-optimized whole-body CT may contribute approximately 10–30 mSv.
2. Consequently, the total exposure from a PET/CT examination may substantially exceed the representative ranges shown in the table.
3. The CT dose report from the individual examination is required to determine the actual CT exposure.

Published data demonstrate substantial interinstitutional and interprotocol variability. Reported mean CT effective doses have been approximately 5 mSv for lower-dose protocols and approximately 15 mSv for diagnostic protocols, with individual diagnostic CT exposures approaching or exceeding 25 mSv.[5–7] One study reported mean CT effective doses of 5.0 mSv with its lower-dose PET/CT technique and 15.4 mSv with diagnostic CT; individual diagnostic CT doses reached 27.8 mSv.

BEIR VII malignancy-risk calculation. BEIR VII Table 12D-1 estimates the following lifetime attributable risks of cancer incidence following a uniform 100-mSv exposure at age 40:

Men: 648 excess cancers per 100,000 exposed persons

Women: 886 excess cancers per 100,000 exposed persons

Linear scaling was applied:

Male excess malignancies per 100,000 = total effective dose in mSv × 6.48

Female excess malignancies per 100,000 = total effective dose in mSv × 8.86

For perspective, the frequently used patient-information estimate that 10 mSv produces approximately one additional malignancy per 1,000 exposed individuals is a reasonable rounded, combined-population approximation. Application of the specific BEIR VII age-40 coefficients gives:

40-year-old man exposed to 10 mSv: approximately 1 additional malignancy per 1,540 persons

40-year-old woman exposed to 10 mSv: approximately 1 additional malignancy per 1,130 persons

These estimates represent excess lifetime cancer incidence, not cancer mortality. They are additional to the underlying spontaneous lifetime incidence of cancer.

§Female risks for prostate-specific radiotracers are included solely to illustrate the sex-associated difference in modeled radiation

Citation: Semelka RC, Ramalho M. PET tracers: radiation exposure and malignancy risk. *J Cancer Prev Curr Res.* 2026;17(4):122–126.
DOI: 10.15406/jcpcr.2026.17.00619

susceptibility at an equivalent effective dose. These values do not imply an approved or clinically relevant indication for prostate-specific tracers in women.

CT Component of Radiation Exposure

Prior PET/CT dosimetry studies demonstrate that the CT component can vary substantially according to whether a lower-dose registration or attenuation-correction technique or a diagnostic-quality CT protocol is used. In one study, the mean total effective dose was 14.0 mSv for PET/CT using the lower-dose CT technique and 24.4 mSv for PET/CT using diagnostic CT.[7]

A recent two-center prospective study measured chromosomal aberrations in peripheral blood lymphocytes before FDG administration, after FDG administration, and after CT acquisition. FDG administration alone was not associated with a statistically significant increase in detectable chromosomal aberrations. The total change from the pre-FDG baseline to completion of PET/CT was significant with standard-dose CT but not with reduced-dose CT, and CT effective dose was independently associated with chromosomal-aberration frequency.[8]

The absence of a statistically detectable increase after FDG administration should not be interpreted as proof that FDG produced no radiation-associated biological injury. FDG contributed a median effective dose of approximately 4.2–4.7 mSv in the study and therefore represented a meaningful baseline radiation exposure. The direct change between the post-FDG and post-CT measurements was not independently significant, whereas the complete pre-FDG to post-PET/CT change was significant with the standard-dose protocol. Standard-dose CT increased the cumulative radiation exposure sufficiently for chromosomal injury to become detectable, while FDG alone may have produced an effect below the study's detection threshold.

Protocol planning must also consider whether a separate diagnostic CT examination will subsequently be required for higher-quality anatomical display. When technically and clinically feasible, a single CT acquisition performed as part of the PET/CT examination and designed to provide attenuation correction, anatomical localization, and the required diagnostic information may be preferable to low-dose PET/CT followed by a separate diagnostic CT. The latter approach may produce partially redundant CT exposure, increased total radiation risk, and no corresponding diagnostic benefit from the duplicated component.

The issue is nevertheless complex. Intravenous contrast timing, respiratory phase, scan coverage, scanner capabilities, image quality, lesion characterization, and the specific clinical question may require a separate diagnostic CT examination. In some circumstances, the radiation dose from an integrated diagnostic PET/CT may also be greater than that from a separately optimized CT protocol. The anticipated entire imaging pathway should therefore be considered before the PET/CT protocol is selected, rather than treating the PET, attenuation-correction CT, and any subsequent diagnostic CT as unrelated examinations.[7,9]

Summation of radiation exposures with repeat PET studies

Patients with cancer may undergo repeated PET/CT examinations at intervals of 3–6 months, and sometimes at shorter intervals. This creates two related but distinct questions:

1. How should the numerical radiation doses be combined?
2. How does the interval between exposures modify the biological and carcinogenic effect?

For radiation-protection purposes, the effective doses from separate examinations may be arithmetically summed to describe cumulative effective dose. For individualized risk estimation, however, it is preferable to treat each examination as a separate exposure event and apply organ-specific dose, sex, age at exposure, latency, attained age, and remaining lifespan. The National Cancer Institute Radiation Risk Assessment Tool permits multiple exposure events to be entered separately and then calculates their combined lifetime risk.[2]

This approach means that the lifetime risk created by the first examination does not disappear before the second examination occurs. Acute DNA injury may be repaired, and measurable DNA-damage markers may decline, but surviving misrepair, genomic alteration, or initiated cellular change may retain long-term significance. The second examination therefore adds radiation exposure and modeled risk to the risk already created by the first.

For example, using the age-40 coefficients employed in the table, two examinations of 10 mSv each performed 1 month apart would be treated conventionally as approximately 20 mSv of cumulative exposure. The estimated combined excess malignancy risk would be approximately:

130 per 100,000 men, or approximately 1 in 770

177 per 100,000 women, or approximately 1 in 560

It is therefore incomplete to describe the second examination only as another isolated "1 in 1,000" risk without also communicating that the two examinations together produce approximately twice the modeled risk of one examination.

If the same two examinations were performed 2 years apart, the first examination would still contribute to lifetime risk. The second examination would be calculated at the later age of exposure and might therefore have a somewhat different lifetime attributable risk, but the earlier 10-mSv exposure would not be erased or discounted simply because 2 years had elapsed. In conventional BEIR VII–based modeling, the combined risk would remain close to that associated with 20 mSv, although it would not be numerically identical because age at exposure and remaining lifespan differ.

What remains unresolved is whether the interval itself creates additional biological interaction beyond this approximate additivity. It is biologically plausible that repair, persistent cellular alterations, inflammatory responses, immune surveillance, clonal evolution, and tissue microenvironment effects could modify the response to a later exposure. However, there is no validated human curve—logarithmic or otherwise—that allows a PET/CT performed 1 week, 1 month, 3 months, or 2 years after an earlier examination to be assigned a specific interval-dependent multiplier.

Experimental split-dose studies demonstrate that the timing and fractionation of radiation can affect measurable DNA-damage responses. Some have shown less detectable damage when a total CT-like dose is divided into fractions, consistent with repair occurring between exposures. These observations do not establish that closely spaced diagnostic examinations produce a superadditive cancer risk, nor do they provide numerical lifetime-risk coefficients applicable to patients.[10]

Citation: Semelka RC, Ramalho M. PET tracers: radiation exposure and malignancy risk. *J Cancer Prev Curr Res.* 2026;17(4):122–126.
DOI: 10.15406/jcpcr.2026.17.00619

Clinical studies of recurrent CT have generally estimated cumulative risk by summing examination doses and applying BEIR VII coefficients according to sex and age at each exposure. In one large adult cohort, 15% of patients accumulated more than 100 mSv from CT, and 7% had a modeled lifetime attributable cancer-incidence risk greater than 1%.[11]

The potential magnitude is readily illustrated for repeated FDG PET/CT. Four examinations with representative total exposures of 12–17 mSv, performed at 3-month intervals over 1 year, would produce an arithmetic cumulative effective dose of approximately 48–68 mSv. Using the same age-40 linear scaling applied in the table, this would correspond to:

Approximately 311–441 excess malignancies per 100,000 men, or approximately 1 in 320 to 1 in 230

Approximately 425–602 excess malignancies per 100,000 women, or approximately 1 in 235 to 1 in 165

These are screening-level modeled estimates rather than individualized predictions. Nevertheless, they demonstrate that serial PET/CT should not be discussed as a succession of unrelated low-risk events. The clinically relevant exposure is cumulative, even though the precise biological relationship among the separate events is not known.

There is an unavoidable clinical irony in using examinations that carry a modeled carcinogenic risk to evaluate individuals who already have cancer. Their biology has already demonstrated the capacity for malignant transformation. This does not establish that every patient with cancer has generalized radiosensitivity, and it does not argue against a medically necessary examination. It does strengthen the case for particular care in younger patients, patients with hereditary cancer susceptibility, patients receiving other carcinogenic therapies, and long-term cancer survivors who may undergo many examinations over their remaining lifespan.

The exact biological effect of cumulative radiation exposures from repeat studies, including whether and how the interval between examinations modifies risk, is unknown at present but is a very important issue that needs to be understood.

Cancer endpoints

The BEIR VII "all cancers" endpoint includes: solid malignancies and leukemia

Leukemia is itself a malignancy and should not be described as a noncancer outcome. Nonmalignant radiation effects are not included in the tabulated risk estimates. At the radiation exposures ordinarily encountered in diagnostic PET/CT, the principal modeled concern is stochastic carcinogenesis rather than deterministic tissue injury.

Important limitations

Effective dose is a radiation-protection quantity developed for comparisons among procedures and populations. It is not a patient-specific measurement of organ dose. The present calculations therefore provide standardized, screening-level estimates rather than individualized predictions.

Individual risk may differ according to:

Age at each exposure

Sex

Organs included in the CT scan

Organ-specific absorbed dose

Body size and anatomy

Underlying disease

Genetic susceptibility

Previous and subsequent radiation exposure

Previous chemotherapy or radiotherapy

Radiotracer kinetics

CT equipment and protocol optimization

Expected lifespan

Number and timing of repeat examinations

Linear scaling of effective dose is useful for illustrating approximate population-level risk, but it cannot reproduce the biological complexity of repeated nonuniform organ exposures. In particular, no validated clinical method currently determines whether examinations separated by short intervals are additive, subadditive, or superadditive at the level of individual carcinogenesis.

Because the CT component can vary more than the radiopharmaceutical component, a meaningful informed-consent discussion should ideally include the anticipated CT technique, the possibility of additional diagnostic CT imaging, the expected frequency of follow-up examinations, and an estimated cumulative effective-dose range rather than quoting the PET tracer exposure alone.[12–21]

Conclusions

PET/CT examinations deliver radiation from both the administered radiotracer and the CT acquisition. Although the PET component is relatively predictable, the CT contribution may vary several-fold and may equal or exceed the radiotracer dose. The selection of reduced-dose versus diagnostic-quality CT therefore materially affects total exposure.

Recent chromosomal-aberration data provide biological support for CT dose optimization but should not be interpreted as demonstrating that FDG produces no biological injury. Rather, the FDG-related effect may have.

remained below the detection threshold, while the greater cumulative exposure associated with standard-dose CT produced detectable chromosomal injury.

Repeated PET/CT examinations may generate substantial cumulative exposure. Current models retain the risk from each prior exposure and combine event-specific estimates, but they do not provide a validated interval-dependent curve describing the interaction of examinations separated by weeks, months, or years. Decisions regarding PET/CT should therefore consider the complete anticipated imaging pathway, including possible additional CT examinations and the cumulative exposure expected from serial follow-up.

Finally, as with everything in medicine a carefully considered risk/benefit analysis should always be made when decisions are made regarding health care management. The risk of the small but real risk of radiation induced malignancy balanced against the benefit to patient care and outcome with the presence of the test information. These

Citation: Semelka RC, Ramalho M. PET tracers: radiation exposure and malignancy risk. *J Cancer Prev Curr Res.* 2026;17(4):122–126.
DOI: 10.15406/jcpcr.2026.17.00619

judgments are generally made on an individual basis. The greater the seriousness of the disease and the evidence-base of the value of the test greatly leans toward acquiring the test. However evaluating conditions which are essentially nonmalignant, the value added by the test may not justify the risk. None the less, the patient should be an active participant in these discussions, and it is appropriate to describe the risk of these procedures, and it can be emphasized that the risk is small compared to the benefit.

Acknowledgement

None.

Conflict of interest

None declared.

References

1. National research council. Health risks from exposure to low levels of ionizing radiation: BEIR VII phase 2. Washington, DC: National Academies Press; 2006.
2. Berrington GA, Apostoaei AI, Veiga LHS, et al. RadRAT: a radiation risk assessment tool for lifetime cancer risk projection. J Radiol Prot. 2012;32(3):205–222.
3. Boellaard R, Delgado BR, Oyen WJG, et al. FDG PET/CT: EANM procedure guidelines for tumour imaging: version 2.0. Eur J Nucl Med Mol Imaging. 2015;42(2):328–354.
4. US Food and Drug Administration. What are the radiation risks from CT? Accessed July 24, 2026.
5. International atomic energy agency. Radiation protection of patients during PET/CT scanning. Accessed July 24, 2026.
6. Jallow N, Christian P, Sunderland J, et al. Diagnostic reference levels of CT radiation dose in whole-body PET/CT. J Nucl Med. 2016;57(2):238–241.
7. Quinn B, Dauer Z, Pandit TN, et al. Radiation dosimetry of ^{2}F-FDG PET/CT: incorporating exam-specific parameters in dose estimates. BMC Med Imaging. 2016;16:41.
8. Kawashima Y, Kinugasa Y, Ishibashi M, et al. Biological effects of ^{18}F-FDG administration and CT dose during PET/CT: chromosomal aberrations in a two-center prospective observational study. Eur Radiol. 2026.
9. International commission on radiological protection. Optimisation of radiological protection in digital radiology techniques for medical imaging. ICRP Publication 154. Ann ICRP. 2023;52(3).
10. Elgart SR, Bostani M, Mok KC, et al. Investigation of DNA damage dose-response kinetics after ionizing radiation schemes similar to CT protocols. Radiat Res. 2015;183(6):701–707.
11. Sodickson A, Baeyens PF, Andriole KP, et al. Recurrent CT, cumulative radiation exposure, and associated radiation-induced cancer risks from CT of adults. Radiology. 2009;251(1):175–184.
12. National library of medicine. Pylarify (piflufolastat F 18 injection): US prescribing information. DailyMed. Accessed June 23, 2026.
13. National library of medicine. Locametz (kit for the preparation of gallium Ga 68 gozetotide injection): US prescribing information. DailyMed. Accessed June 23, 2026.
14. National library of medicine. Posluma (flotufolastat F 18 injection): US prescribing information. DailyMed. Accessed June 23, 2026.
15. National library of medicine. Axumin (fluciclovine F 18 injection): US prescribing information. DailyMed. Accessed June 23, 2026.
16. National library of medicine. Cerianna (fluoroestradiol F 18 injection): US prescribing information. DailyMed. Accessed June 23, 2026.
17. National library of medicine. Amyvid (florbetapir F 18 injection): US prescribing information. DailyMed. Accessed June 23, 2026.
18. National library of medicine. Neuraceq (florbetaben F 18 injection): US prescribing information. DailyMed. Accessed June 23, 2026.
19. National library of medicine. Vizamyl (flutemetamol F 18 injection): US prescribing information. DailyMed. Accessed June 23, 2026.
20. National library of medicine. Tauvid (flortaucipir F 18 injection): US prescribing information. DailyMed. Accessed June 23, 2026.
21. National library of medicine. Netspot (kit for the preparation of gallium Ga 68 dotatate injection): US prescribing information. DailyMed. Accessed June 23, 2026.