showing high uptake of ^{201}Tl. I would like to point out that the scan was performed only 10 min after injection of thallium and no further delayed imaging was performed. According to our own experience as well as past reports, the early uptake of thallium was relatively nonspecific, since pulmonary edema and hyperemia associated with infections were known to cause high thallium uptake (*2–4*). In contrast to neoplastic uptake, pulmonary thallium uptake in infected foci washed out relatively fast, while that of tumors remained high relative to background (*2–4*). The characteristic tracer dynamics enabled us to differentiate AIDS-related Kaposi's sarcoma from other common chest infections among immunodeficient patients by performing delayed (3 hr) thallium scans (*2*). Similar technique of using delayed thallium scans was also reported in the past to differentiate neoplasms from other benign lesions in the lung and thyroid (*3–5*). One notable exception to this rule was reported by Tonami et al. who demonstrated that there was persistent thallium uptake in a single case of cerebral candidiasis in their delayed scans (*6*). It is still unclear whether this represented alteration of tracer kinetics because the lesion was located in the brain instead of lung. It is interesting to note a separate report by the same authors that the thallium washout rate of tumors was very different in the breast and lungs. They concluded that to evaluate lung lesions using thallium scintigraphy delayed scans had to be performed to obtain optimal results (*6*).

One of the advantages of using thallium instead of gallium for tumor imaging is that we can differentiate between infectious and neoplastic lesions using thallium. Gallium is avid in both. Therefore, the importance of performing delayed thallium scan cannot be overemphasized.

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REPLY: We appreciate very much the comments of Dr. Victor W. Lee on our recently published article (*1*). In order to get a more objective conclusion from our report, some additional aspects of ^{201}Tl imaging for detecting inflammatory focus and tumor should be emphasized.

1. Dr. Lee pointed out that ^{201}Tl imaging 10 min after injection was too early to evaluate the uptake by a pulmonary lesion and suggested that tumors could be differentiated from inflammatory lesions through delayed scans. However, it was shown that ^{201}Tl uptake by tumors occurred rapidly, the mean time from injection to peak tumor activity being 11.9 min for lung carcinoma, 11.2 min for breast carcinoma and 11.7 min for lymphoma (*2–3*). In another study, the accumulation ratio between the tumor and the contralateral normal lung reached its highest level between 30 and 60 min (*4*). In addition, several reports on clinical application showed that early scans obtained 1–20 min after the injection could localize malignant tumors successfully (*5–8*). In an experimental study, Ando et al. showed that in inflammatory lesions in addition to tumor tissue quite large amounts of ^{201}Tl accumulated in subcutaneous tissue infiltrated with neutrophils and macrophages, regardless of the time after administration (*9*). Thallium-201 accumulation in tumors was found to decrease gradually 1 hr after administration (*10*). These reports reveal that it is very difficult to differentiate malignant tumors from inflammatory lesions through delayed scans.

2. Although we showed no definite mechanism to explain the unexpected uptake of ^{201}Tl by pulmonary actinomycosis in our case report, this uptake, without any experimental findings, cannot be simply attributed to pulmonary edema and hyperemia. Increased blood flow, hypervascularity, hyperpermeability, increased membrane-pore size, Na^+-K^+ ATP-ase dependent pump activity, increased cellular K^+ content and metabolism should also be considered in the uptake of ^{201}Tl by tumors and inflammatory lesions.

3. In our case of pulmonary actinomycosis, we initiated ^{201}Tl imaging 10 min after injection and obtained spot, whole-body and SPECT images, all of which normally take 70–80 min. After examining all of the images, we did not find any difference of note in the activity accumulated in the inflammatory lesion.

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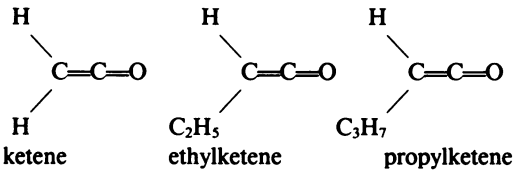
Incorrect Naming of a Carbon-11-Labeled Reagent

TO THE EDITOR: I would like to point out a trivial, but perhaps important, error in the paper entitled "No-Carrier-Added Carbon-11-Labeled sn-1,2- and sn-1,3- Diacylglycerols by [^{11}C] Propyl Ketene Method" published in the *Journal* (*J Nucl Med* 1991;32:1622–1626). The error is in the naming of the reagent in the title and throughout the text. The authors have called the ketene formed propyl ketene, but it is in fact ethylketene. The structure that they depict in Figure 1 is correct, but the name is incorrect. Structures of ketenes are as follows, and can be obtained from general organic textbooks.



I pointed out the fact that the nomenclature for this compound was incorrect when it was presented at the 8th International Symposium on Radiopharmaceutical Chemistry (*J Lab Compds Radiopharm* 1991;30:127–128), but apparently the authors did not understand. I am concerned that a trivial error like this will be propagated further in the literature unless a correction is made in your journal.

If you have questions about my concerns on nomenclature, please ask one of the chemists on your Editorial Board to review this issue. Thank you for your efforts.

D. Scott Wilbur
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REPLY: We thank Dr. Wilbur for communicating with us concerning the naming problem of alkylketene compounds (*1*). $C_2H_5CH=^{11}C=O$ should be named ^{11}C-labeled ethylketene. However, the inappropriate naming in the article does not affect our conclusions concerning the ability of the new labeling method using ketene reaction. Our experiments have shown that several ^{11}C-labeled alkylketenes can be formed in the same condition as described in the article. For example, $^{11}CO_2$ reacted to the alkyl lithium mixture which consisted of the same equivalent of *n*-propyl lithium (2.2 μmol) and *n*-butyl lithium (2.2 μmol). Carbon-11-labeled ethylketene and propylketene were formed from *n*-butyric acid and *n*-valeric acid by the pyrolytic decomposition, respectively, as follows:

$$C_3H_7Br + 2\,Li \rightarrow C_3H_7Li + LiBr$$
n-propyl bromide — *n*-propyl lithium

$$^{11}CO_2 + C_3H_7Li \rightarrow C_3H_7{}^{11}COOH \xrightarrow{HCl} C_2H_5CH=^{11}C=O$$
[1-^{11}C]butyric acid 530°C [^{11}C]ethylketene

$$C_4H_9Br + 2\,Li \rightarrow C_4H_9Li + LiBr$$
n-butyl bromide — *n*-butyl lithium

$$^{11}CO_2 + C_4H_9Li \rightarrow C_4H_9{}^{11}COOH \xrightarrow{HCl} C_3H_7CH=^{11}C=O$$
[1-^{11}C]valeric acid 530°C [^{11}C]propylketene

Generally, ketene is an extremely unstable compound so that we could not detect any ketenes as naturally active molecules (*2*). However, we easily obtained the acylated compound as [^{11}C] alkylketene adduct (*3*). These ^{11}C-labeled alkylketenes produced the simultaneous formation of *rac*-1,2,-[^{11}C]diacylglycerols, 1-[1-^{11}C]butyryl-2-palmitoyl-*rac*-glycerol and 1-[1-^{11}C]valeryl-2-palmitoyl-*rac*-glycerol as shown in Figure 1. This suggests the equality of producing [^{11}C]alkylketene formation in smaller degrees of alkyl carbon chains.

We believe that the ketene reaction could be more general and not be necessarily limited to [^{11}C]propylketene or [^{11}C]ethylketene because another alkylketene, which has smaller alkyl carbon chains, can also be produced by the same procedures. We believe this is a good opportunity to define the "Ketene Method" as an all inclusive term.

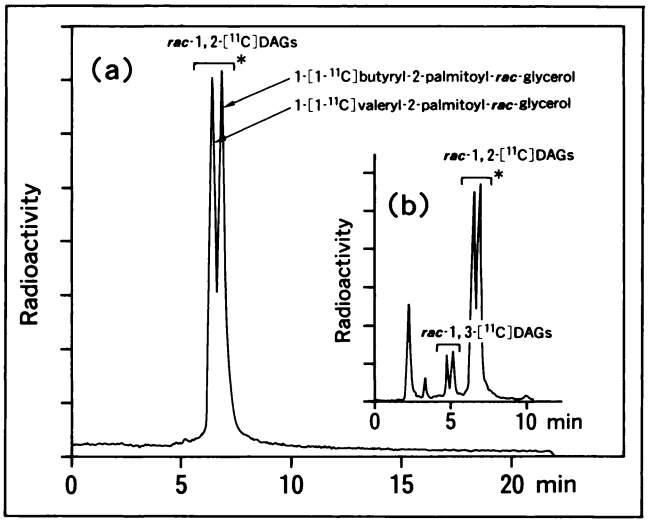

FIGURE 1. Radio-HPLC profile of *rac*-1,2-[^{11}C]diacylglycerols (a) separated from [^{11}C]alkylketene adducts (b). [^{11}C]alkylketenes, [^{11}C]ethylketene and [^{11}C]propylketene formed from n-[1-^{11}C]butyric acid and *n*-[1-^{11}C]valeric acid, respectively, react to 2-palmitoylglycerol. Zorbax SIL (DuPont Instrument, 4.6 mm × 25 cm) was used for the analysis of [^{11}C]alkylketene adducts. HPLC was performed at room temperature, and *rac*-1,2-diacylglycerols (*rac*-1,2-DAGs) were separated by using hexane-isopropyl alcohol (194:6 v/v). The flow rate was 1.8 ml/min. (a) The simultaneous formation of *rac*-1,2-[^{11}C]DAGs, 1-[1-^{11}C]butyryl-2-palmitoyl-*rac*-glycerol (6.8 min) and 1-[1-^{11}C]valeryl-2-palmitoyl-*rac*-glycerol (6.4 min). Time means the retention time on HPLC analysis. (b) [^{11}C]alkylketene adducts and inpurities before the HPLC separation.

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The Importance of a Delayed Scan in Thallium Imaging––Reply

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