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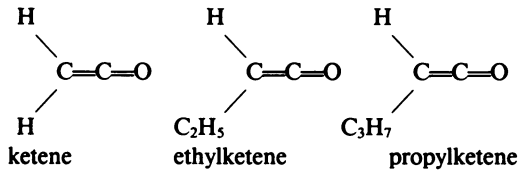
Incorrect Naming of a Carbon-11-Labeled Reagent

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.

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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.

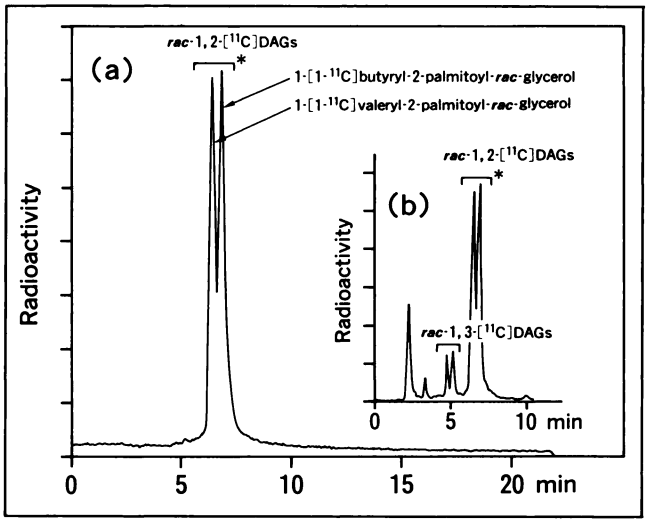

FIGURE 1. Radio-HPLC profile of *rac*-1,2-[^{11}C]diacylglycerols (a) separated from [^{11}C]alkylketene adducts (b). [^{11}C]alkylketenes, [^{11}C]ethylketene and [^{11}C]propylketene formed from n-[1-^{11}C]butyric acid and *n*-[1-^{11}C]valeric acid, respectively, react to 2-palmitoylglycerol. Zorbax 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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Proper Definitions for Lag Phase in Gastric Emptying of Solid Foods

TO THE EDITOR: Considerable controversy and confusion exists over the proper definition of the "lag phase" associated with the gastric emptying of solid foods. We are concerned that the conclusions reported in the recent publication by Christian et al. (*1*) may only add to this confusion.

The authors reported "a short solid meal lag phase (average 8.6 min) exists that can be missed with conventional radionuclide gastric emptying methods not employing continuous measurements." While we find no fault with the authors' experimental results, we believe that their finding of such a short lag phase is the result of failure to use an adequate solid radiolabeled meal. Most importantly, their conclusion that continous measurements are needed to perform a gastric emptying study is incorrect and, if accepted, would needlessly increase the complexity of this study.

Cannon first observed that the fundus and antrum play separate roles in emptying liquids and solids (*2*). He proposed that the fundus acts as a reservoir which initially undergoes receptive relaxation to receive food from the esophagus (*3*). Solids are then moved from the fundus to the antrum. Once in the antrum large particulate solids are ground by antral contractions into smaller particles by a process termed trituration. As stated correctly by Christian et al., solid particles do not empty through the pylorus until they are reduced to particles 1–2 mm in size (*4,5*).

We believe any definition of the lag phase must reflect the known physiology of gastric emptying of solids. We have previously shown that a lag phase based upon a definition that includes time for receptive relaxation and trituration is a function of ingested particle size and meal composition (*6,7*). Such a measure of the lag phase using the modified power exponential function has been studied using geometric mean attenuation correction and correlates with peak antral filling (*7*). This suggests that once solids fill the atrum and have been adequately triturated they begin to empty.

In their article, Christian et al. fail to ascribe any physiologic significance to their very short lag phase. We believe the very short lag phase reported by Christian et al. is merely a result of the fact that they have not used a sufficiently solid labeled test meal. They have labeled a liver pate, which as they state in their Material and Methods section, consists of particles 2–5 mm in size. Most of these particles therefore do not need to undergo trituration. Their short lag phase likely represents only the time for these small particles to reach the antrum following which they quickly begin to empty. The true solid in their study was the beef stew which was not labeled and therefore not evaluated.

In an earlier article, these authors validated their surface-labeled pate by comparison to an intracellular label and found similar emptying curves. It should be noted however that they diced the intracellularly-labeled chicken liver cubes into "2–3 mm chunks" (*8*).

As pointed out by Christian et al., numerous recent papers have reported that a lag phase for solid food emptying does exist. All these reports have used either whole in vivo chicken liver or labeled egg and not a liver pate (*7–14*).

Both articles they cited, which have supported the concept of no lag phase, used a radiolabel that did not label the solid [e.g., technetium-sulfur colloid mixed with mashed potato (*15*) and chromium-51 in porridge (*16*)]. In those articles, in which a true radiolabeled solid was employed, the graphs presented all show clear evidence of a lag phase (*5,17*).

The best definition of the lag phase remains controversial. Some have chosen to define the lag phase visually as "the part of the solid-emptying curve prior to the appearance of detectable amounts of radiolabel of the solid phase in the proximal small intestine" (*9*). While there is currently no consensus on the best method to measure the lag phase, it does appear to have clinical significance. It is a sensitive indicator of drug interventions employed to treat diabetic gastroparesis (*18*). Analysis of the lag phase has also been used to study the effects of ulcer surgery on gastric emptying. Mayer found obliteration of the lag phase following truncal vagotomy and pyloroplasty without an effect on trituration (*13*).

We believe the lag phase is best defined using a mathematical definition based on a model such as the modified power exponential curve fit (*6,7*). This obviates the need for continuous image acquisition using dual detectors which is not practical. Before committing others to such an approach, Christian et al. need to justify their conclusions by proposing a physiologic explanation for their very short lag phase.

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