Correction. In the article "Phospholipid methylation in macrophages is inhibited by chemotactic factors" by Marilyn C. Pike, Nicholas M. Kredich, and Ralph Snyderman, which appeared in the June 1979 issue of *Proc. Natl. Acad. Sci. USA* (**76,** 2922–2926), an undetected printer's error occurred in Fig. 7 such that the peaks were incorrectly labeled. The correct figure is shown here.

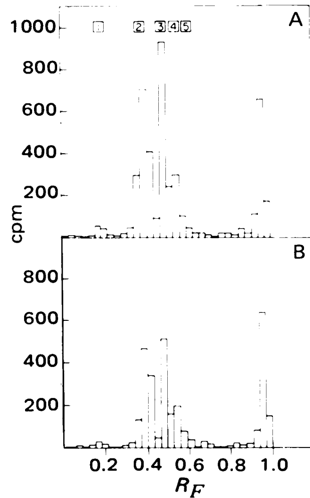

FIG. 7. Chromatographic pattern of the [^{3}H]methylated phospholipid reaction products in the presence of medium alone (*A*) and of 10 nM fMet-Met-Met. Reaction products were isolated, applied to a silica gel G plate, and chromatographed with a solvent system of chloroform/propionic acid/*n*-propyl alcohol/water, 2:2:3:1 (vol/vol). Similar results were obtained with chloroform/methanol/water, 65:25:4 (vol/vol). Region identification: *1*, lysophosphatidylcholine; *2*, PtdCho; *3*, phosphatidyl -*N,N*-dimethylethanolamine; *4*, phosphatidyl -*N*- monomethylethanolamine; *5*, PtdEtn.

Correction. In the article "Isolation of a herpesvirus-specific DNA polymerase from tissues of an American patient with Burkitt lymphoma" by H. S. Allaudeen and J. R. Bertino, which appeared in the September 1978 issue of *Proc. Natl. Acad. Sci. USA* (**75,** 4504–4508), an error occurred in the editorial office. On page 4504, lines 16–19 in the left-hand column should read ". . . The herpesvirus-induced DNA polymerase can be distinguished from cellular DNA polymerases α, β, and γ by differences in their properties (6, 8, 9)."

FIG. 1. (*Left*) Sodium dodecyl sulfate/polyacrylamide gel electrophoresis of iodinated sparse and confluent cultures of bovine aortic endothelial and smooth muscle cells. Washed cells were radiodinated, lysed, and analyzed by gradient (6.5%–16%) gel electrophoresis either before (lanes A–D) or after (lanes E–H) reduction with 0.1 M dithiothreitol. Endothelial cells: Confluent (lanes A and E) and sparse (lanes B and F). Bovine aortic smooth muscle cells: Confluent (lanes C and G) and sparse (lanes D and H). Gels were standardized with T4 phage [^{35}S]methionine-labeled proteins, and arrows mark the positions of fibronectin and CSP-60. (*Right*) Effect of urea and mild trypsinization on the appearance of CSP-60 and fibronectin in confluent monolayers of endothelial cells. Vascular endothelial monolayers, 3 weeks after reaching confluence, were treated with either urea (1 M in Dulbecco's modified Eagle's medium, 1 hr, 37°C) or trypsin (Worthington, 0.2 μg/ml, 1 hr, 37°C), washed, and iodinated. Samples were applied to a gradient (5–16%) polyacrylamide slab gel either before (lanes A–C) or after (lanes D–F) reduction with 0.1 M dithiothreitol. (Lanes A and D) Untreated, highly organized endothelial monolayer. Arrows mark the positions of fibronectin and CSP-60. (Lanes B and E) Confluent endothelial monolayers disrupted by urea into single round cells. Note the absence of CSP-60. The fibronectin of the extracellular matrix becomes exposed for iodination. (Lanes C and F) A confluent cell monolayer iodinated after mild trypsinization, which did not disorganize the monolayer configuration of the culture. Little or no fibronectin is left, but there is no effect on CSP-60. The appearance of CSP-60 in confluent cell monolayers has been demonstrated in all the vascular endothelial lines tested so far, regardless of their origins and ages. This includes cells derived from fetal bovine heart; fetal, calf, and adult bovine aortic arches; bovine pulmonary artery; bovine umbilical vein; and pig aortic arch.

FIG. 4. Appearance of CSP-60 and fibronectin after disorganization and a subsequent reorganization of a confluent cell monolayer. Confluent endothelial monolayers were treated with either trypsin [0.05% (GIBCO) 3 min, 37°C], EDTA (0.03% in Ca^{2+},Mg^{2+}-free phosphate-buffered saline, 30 min, 37°C), or urea (1 M in medium, 1 hr, 37°C) to dissociate cell-to-cell contacts. The disruptive agent was then washed out and the cells were allowed to reorganize in medium containing a fibronectin-depleted serum on the same plate or reseeded at a high or low density. Disorganized and reorganized cultures were iodinated and analyzed by gel electrophoresis (a 6–16% gradient) after reducing the samples with dithiothreitol. Lanes: (A) Confluent endothelial cell monolayer. (B) Confluent culture that was first trypsinized into single cells and then washed and incubated in the same plate for 12 hr under growth conditions to readopt its original monolayer configuration. (C) Cells 12 hr after trypsinization and seeding at a high density. The cells fully adopt a monolayer organization and show the presence of CSP-60, but little or no fibronectin. (D) Cells 12 hr after trypsinization and seeding at a low density. (E) Confluent culture treated with EDTA and labeled when the cells detached from each other. CSP-60 is now only slightly or no longer exposed for iodination. (F) Confluent culture that was first dissociated by EDTA and then incubated for 5 hr under growth conditions to readopt its original morphology. (G) Cells 12 hr after EDTA-dissociation and seeding at a high density. (H) Cells 12 hr after EDTA-dissociation and seeding at a low density. (I) Confluent cell monolayer. (J) Confluent culture treated with urea and labeled when the cells appeared as single round spheres. (K) Confluent culture that was first treated with urea and then allowed to reorganize by a 3-hr incubation under growth conditions. (L) Cells 24 hr after exposure to urea and reseeding at a high density. (M) Cells 24 hr after treatment with urea and reseeding at a low density. Arrows mark the positions of fibronectin and CSP-60.