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Preferential Sequestration of a Vanadyl Chelate by Cancer Tissue Facilitates 2-(Fluorine-18)-2-Deoxyglucose (FDG) Uptake for Positron Emission Tomography (PET) Imaging

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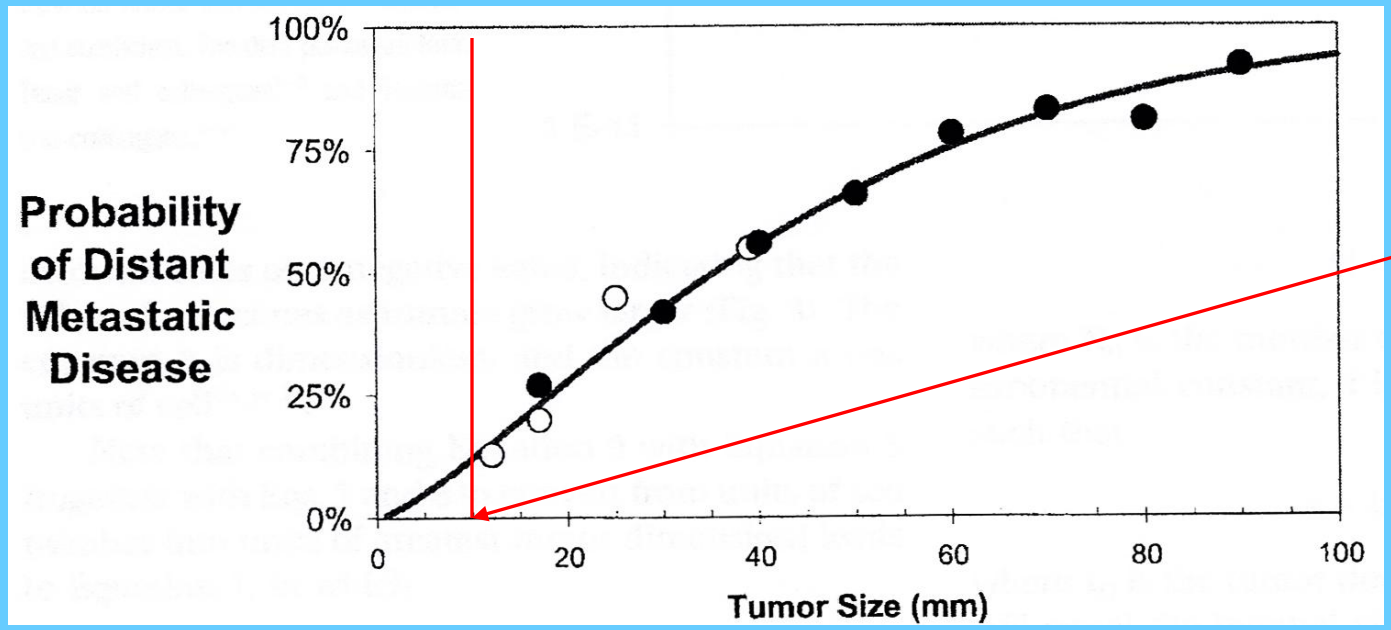
Morbidity and Mortality Statistics of Breast Cancer Disease

FIVE-YEAR SURVIVAL RATES

Absence of metastases at time of detection: 98%

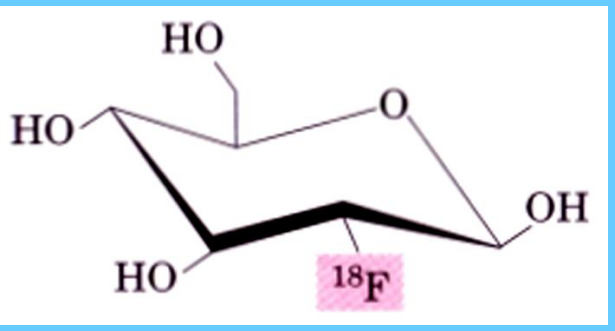
Presence of metastases at time of detection: 23%

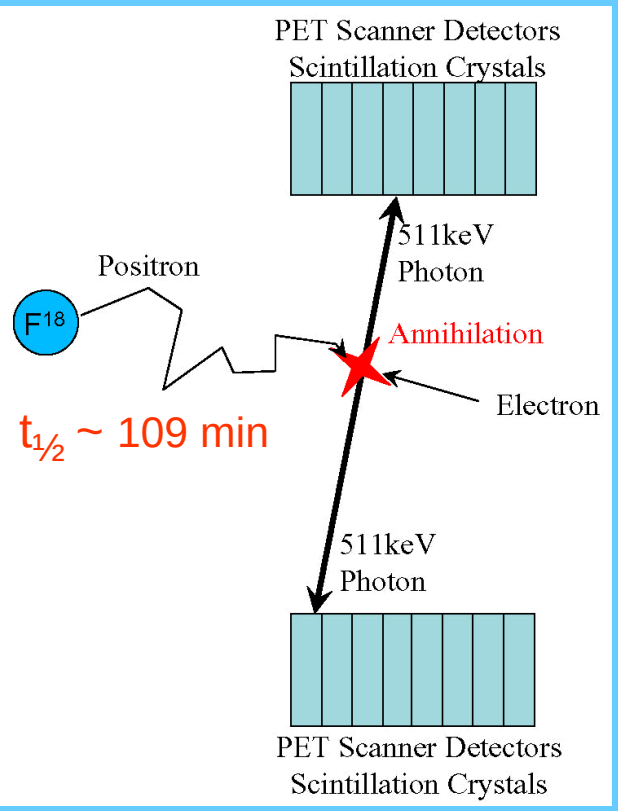
American Cancer Society. (2009) "Cancer Facts & Figures 2009-2010 ". http://www.cancer.org/downloads/STT/F861009_final%209-08-09.pdf.

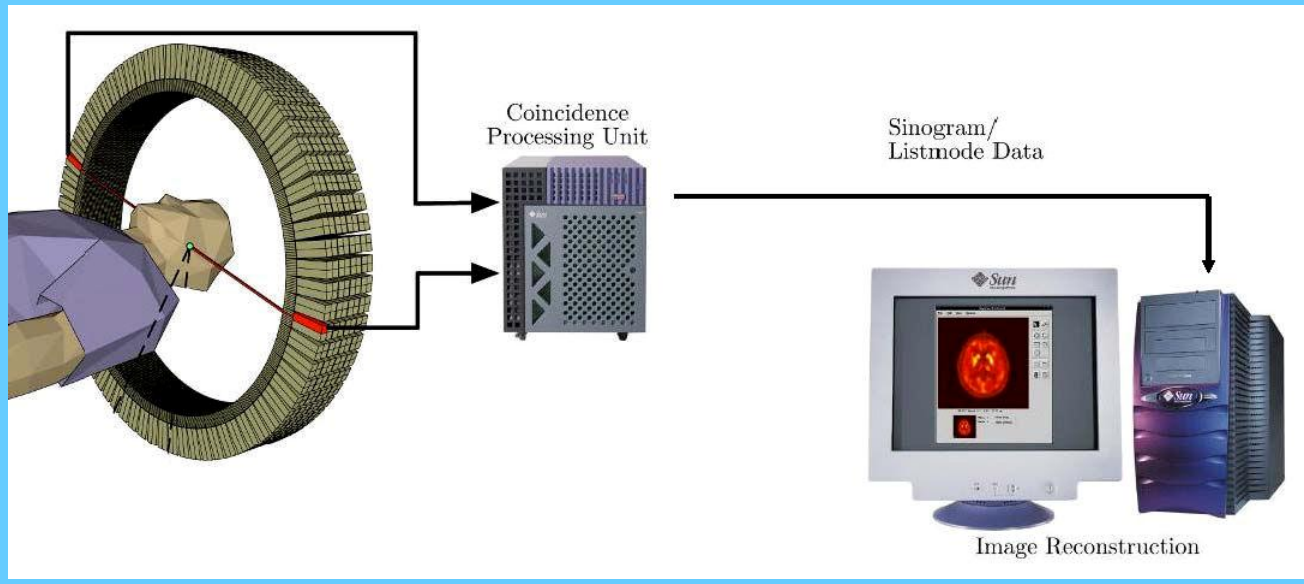


Michaelson et al. *Cancer* 95: 713 – 723 (2002)

Positron Emission Tomography (PET) Imaging with 2-(Fluorine-18)-2-deoxy-D-Glucose

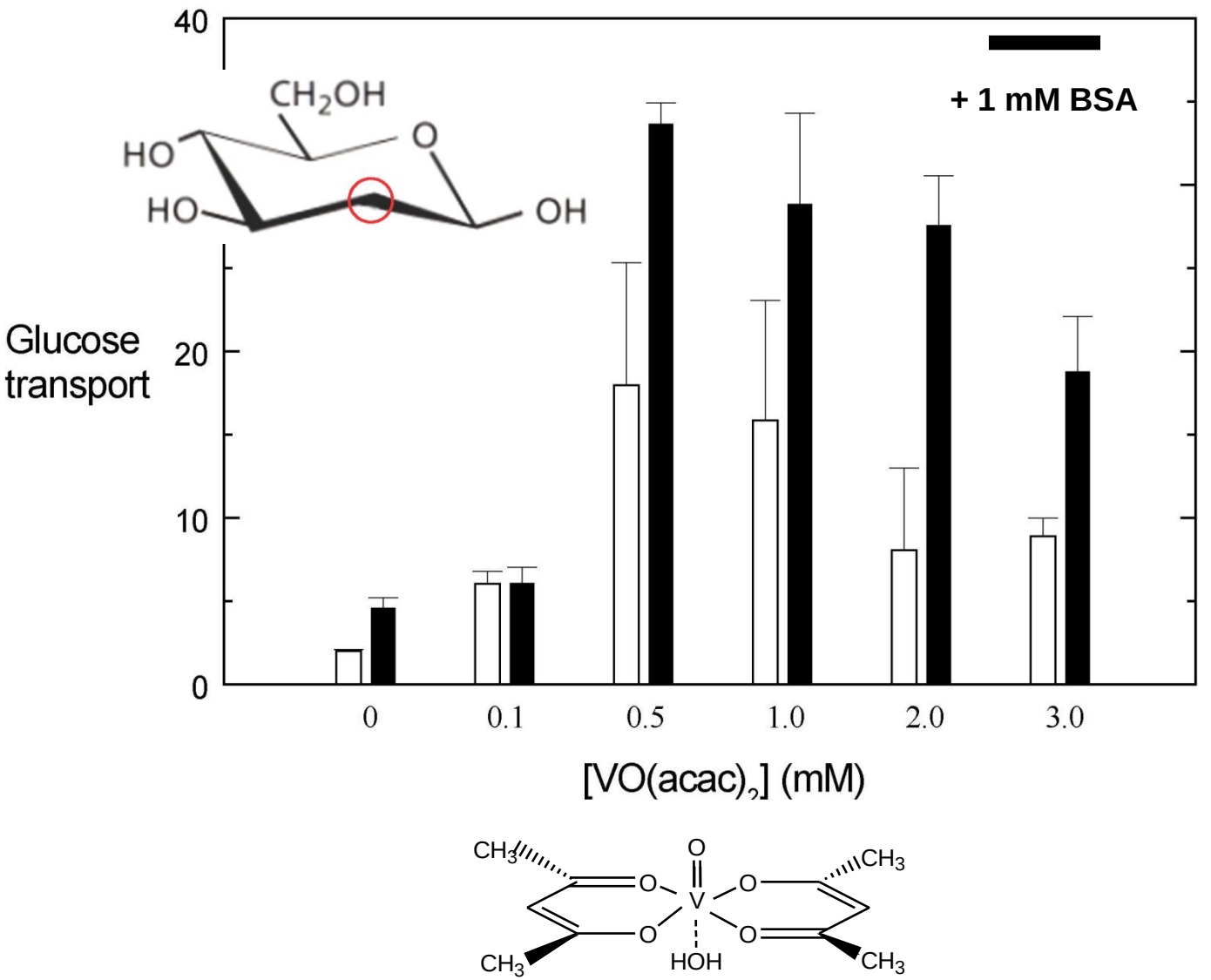






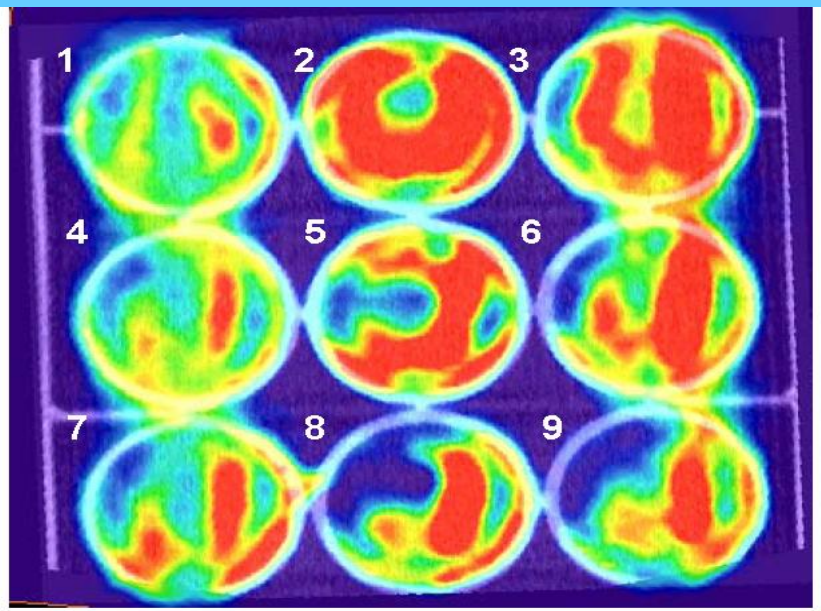
What advantages does FDG PET imaging have over other modalities of cancer detection?

Glucose uptake by cellular systems is commonly measured with use of 1-(C-14)-2-deoxy-D-glucose.



Makinen & Brady, *JBC* 2002, *277*, 12215.

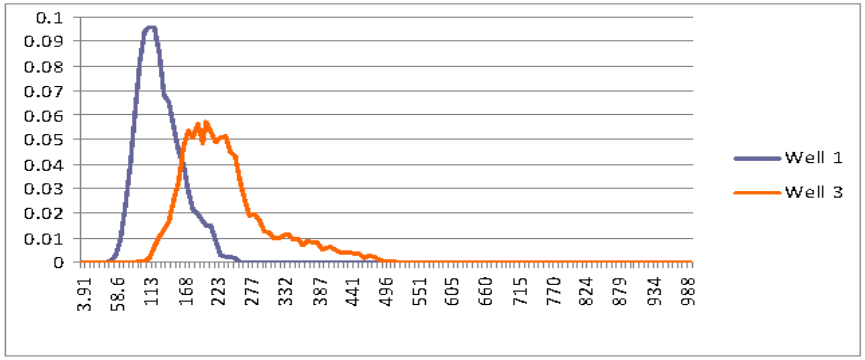
Measurement of $VO(acac)_2$ Facilitated Uptake of FDG by MDA-MB-231 Cells



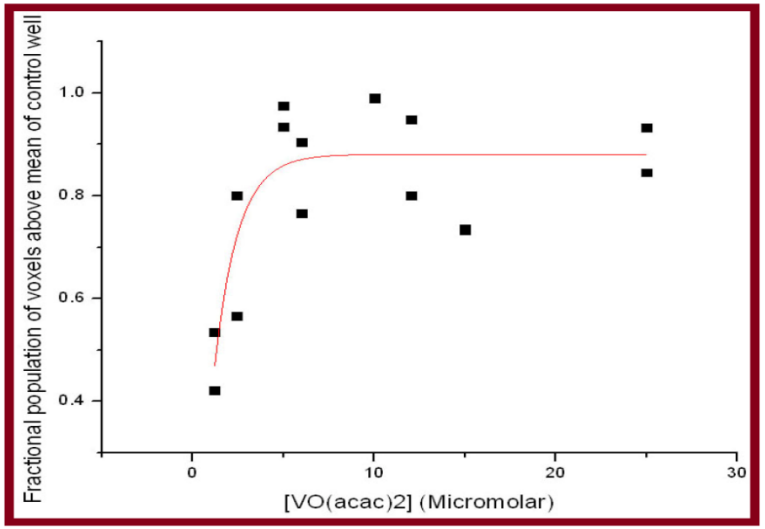
Coronal images of FDG uptake by MDA-MB-231 cells cultured in 9-well plates

Vanadyl concentrations:
Well 1 (0 μM)
Wells 2 and 5 (5 μM)
Wells 3 and 6 (10 μM)
Wells 4 and 7 (15 μM)
Wells 8 and 9 (25 μM).

Color is proportional to uptake of FDG (Blue=least intense; Red=most intense).

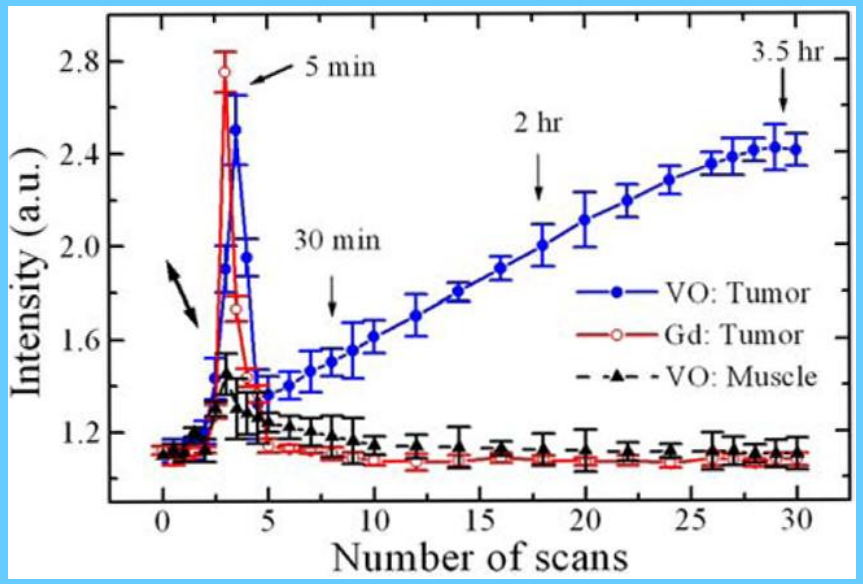


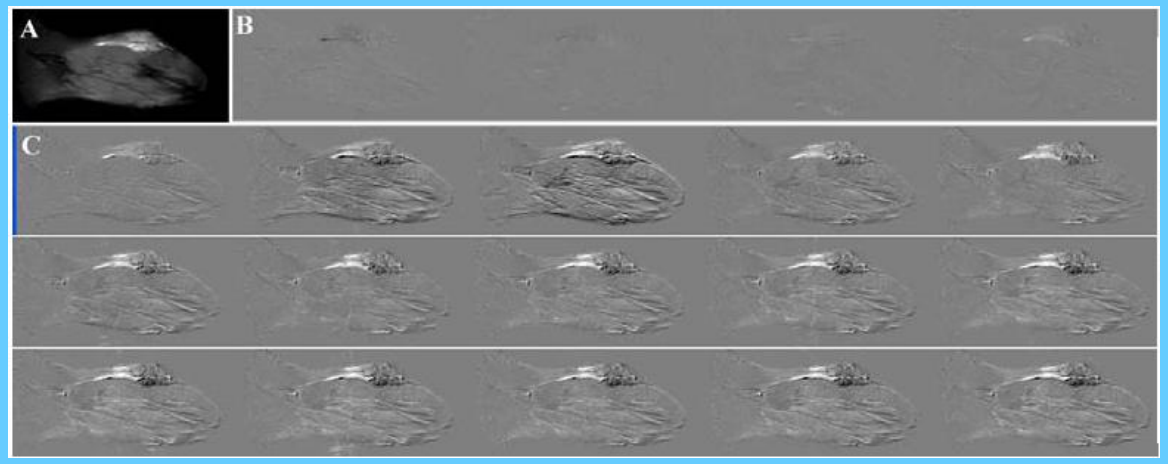
Fraction of Voxels: y-axis
Voxel Intensity: x-axis



The Scatter Plot Depicting the Fraction of Voxels in Wells Containing $VO(acac)_2$ with Intensity above the Mean of the Control Well.

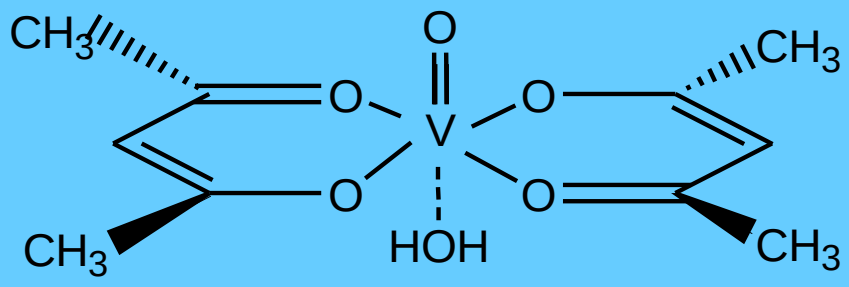
$VO(acac)_2$ is accumulated and retained by tumor tissue significantly longer than by the normal surrounding tissue.

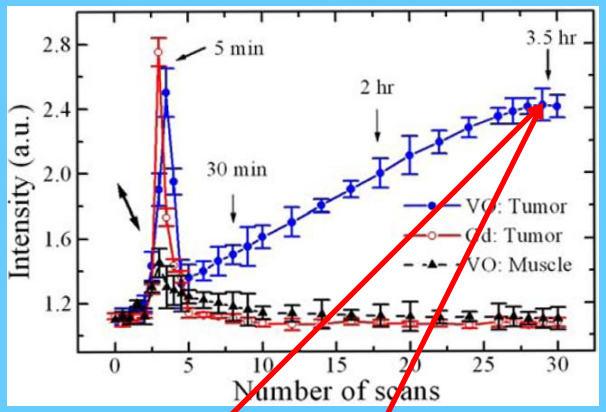


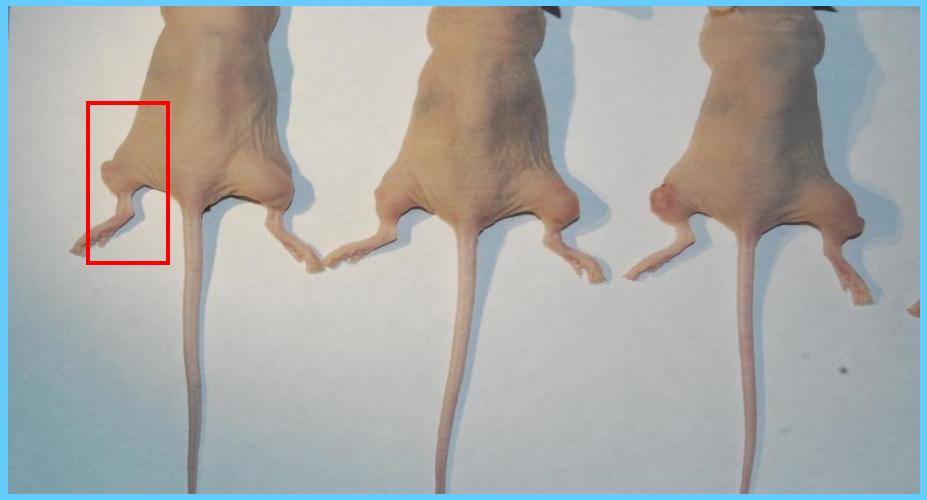


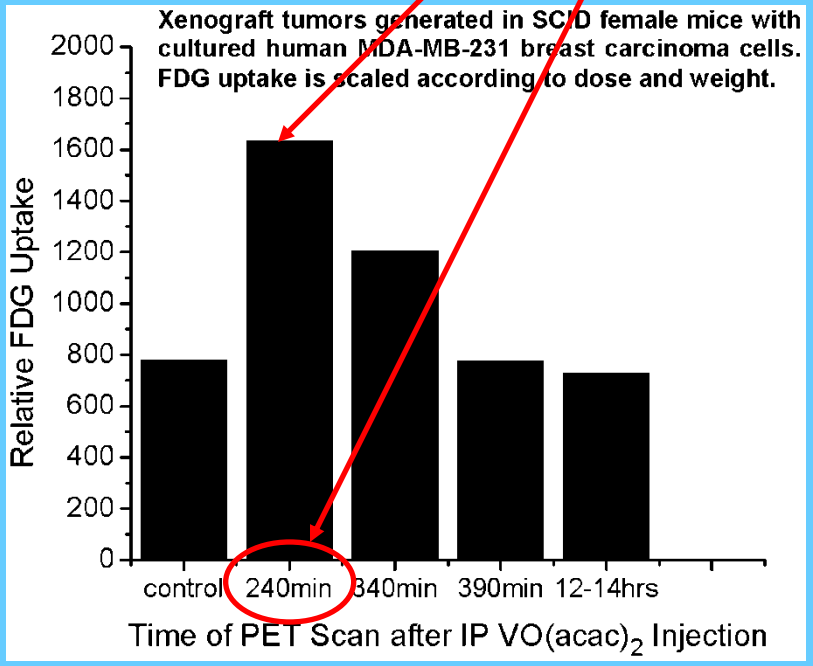
D. Mustafi *et al*., *JBIC* 2009, *14*, 1187-1197.

$VO(acac)_2$ enhances uptake of FDG by xenograft tumor tissue over 2-fold compared to uptake in the absence of the chelate.

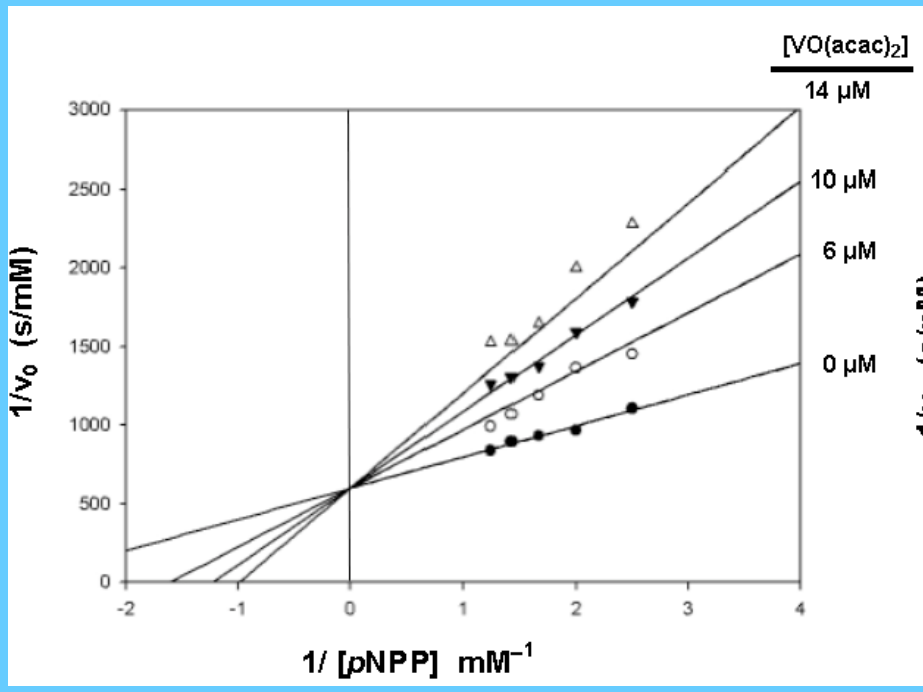


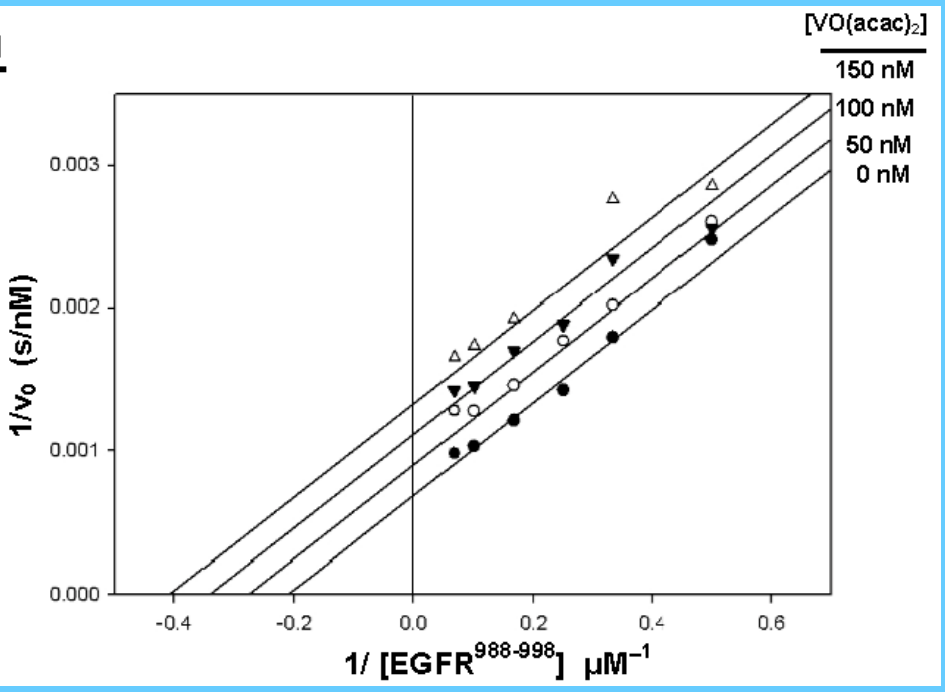




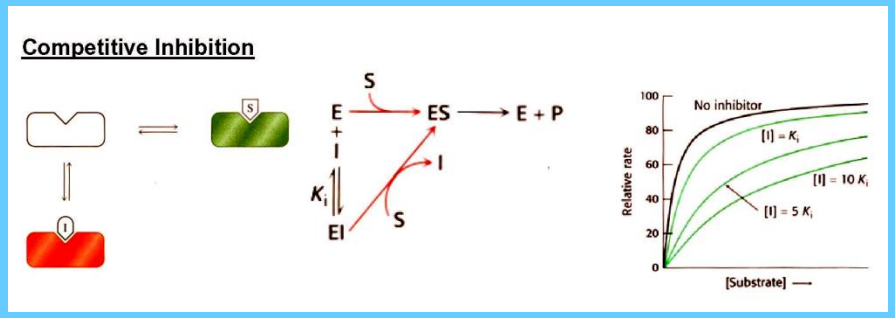


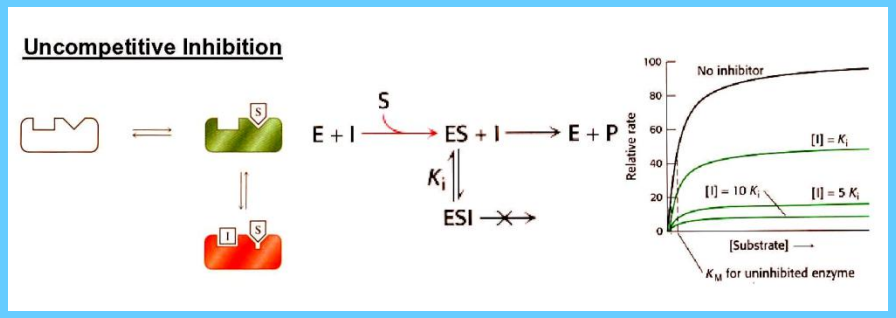
$VO(acac)_2$ is an <u>uncompetitive</u> inhibitor of PTP-1B in the presence of a physiologically relevant substrate. This action hinders dephosphorylation of the IRS mediators important in cell-signaling for glucose uptake.



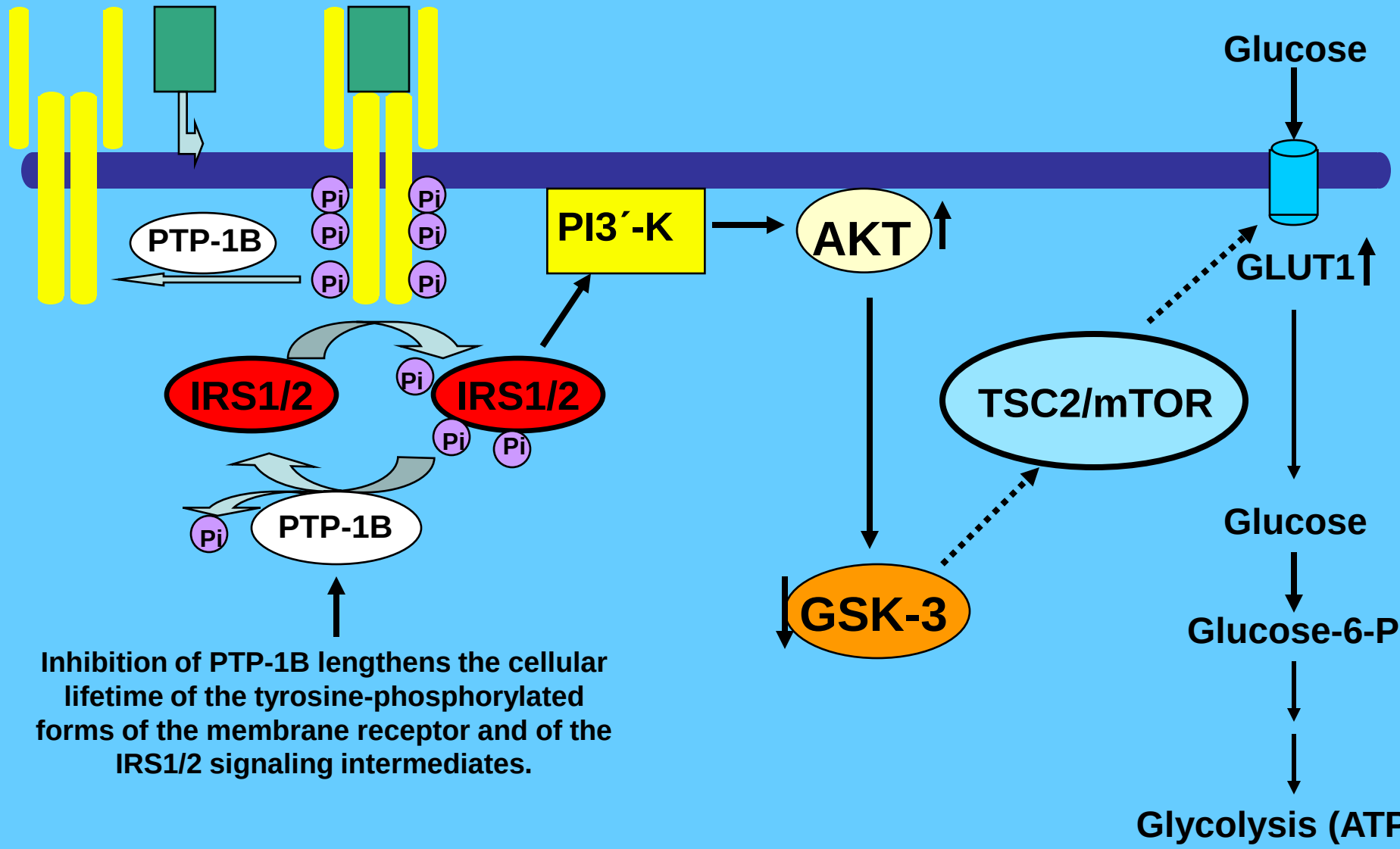


An uncompetitive inhibitor is more potent pharmacologically than a competitive inhibitor in open systems, such as cells, in which there is a continual supply of new substrate.





How does inhibition of PTP-1B by $VO(acac)_2$ enhance glucose uptake by cancer cells?



Inhibition of PTP-1B lengthens the cellular lifetime of the tyrosine-phosphorylated forms of the membrane receptor and of the IRS1/2 signaling intermediates.

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