

Errata

Nucleoside transport inhibitors, dipyridamole and *p*-nitrobenzylthioinosine, selectively potentiate the antitumor activity of NB1011

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The second and third sentences in the Results section of this article¹ should read:

The median-effect/combination index method by Chou and Talalay was used to calculate CI values. CI values <1 indicate synergy, CI=1 indicates additivity and CI>1 indicates antagonism

Reference

1. Boyer RB, Karjian PL, Wahl GM, Pegram M, Neuteboom STC. Nucleoside transport inhibitors, dipyridamole and *p*-nitrobenzylthioinosine, selectively potentiate the antitumor activity of NB1011. *Anti-Cancer Drugs* 2002; 13: 29–36.

The prospects of retinoids in the treatment of prostate cancer

Lisette A Hammond,¹ Geoffrey Brown,² Richard G Keedwell,² Jennifer Durham² and Roshantha AS Chandraratna³

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The following corrections to this article¹ should be noted:

The left-hand panel of Figure 3 should include the symbols for the 2 μ M results as indicated.

A revised Table 1 showing the correct receptor specificities is given overleaf.

Reference

1. Hammond LA, Brown G, Keedwell RG, Durham J, Chandraratna RAS. The prospects of retinoids in the treatment of prostate cancer. *Anti-Cancer Drugs* 2002; 13: 781–90.

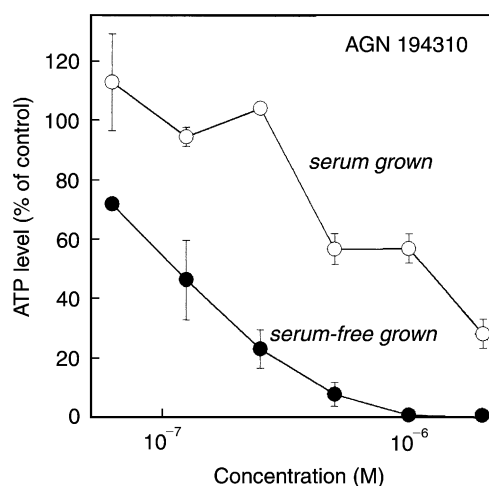


Figure 3. Antagonists are more effective against serum-free grown than serum grown prostate cancer cells.

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Table 1. Novel retinoid analogs, and their receptor binding and transactivation properties [see original article for structures]

Compound no. (AGN)	Receptor specificity ^a	PAR α		PAR β		PAR γ	
		K_d ^b (nM)	EC ₅₀ ^c (nM)	K_d ^b (nM)	EC ₅₀ ^c (nM)	K_d ^b (nM)	EC ₅₀ ^c (nM)
194078	RAR α agonist	4	140	> 5000	WA ^d	> 5000	NA ^d
195153	RAR α agonist	40	130	> 5000	WA ^d	> 5000	WA ^d
190299	RAR $\beta\gamma$ agonist	616	> 1000	41	18	57	42
194310	RAR $\alpha\beta\gamma$ antagonist	3	NA ^d	2	NA ^d	5	NA ^d
193109	RAR $\alpha\beta\gamma$ antagonist	2	NA ^d	2	NA ^d	3	NA ^d
194301	RAR α antagonist	3	NA ^d	320	NA ^d	7250	NA ^d
194431	RAR $\beta\gamma$ antagonist	300	NA ^d	6	NA ^d	70	NA ^d

^aNone of the compounds bound to (K_d values > 10 μ M) or activated any of the RXR subtypes.

^bReceptor binding was determined with full-length, baculovirus-expressed receptors in competitive binding assays using radiolabeled ligands.

^cFunctional activity of the compounds was determined in CV-1 cells transiently transfected with an appropriate RAR/RXR- or RXR/RXR-responsive reporter gene together with an expression vector for a specific receptor subtype.

^dAbbreviations: NA, inactive; WA, weak partial agonist.