



[Update Your Profile](#)

## Jeffrey A. Winkles, PhD

Academic Title:

Professor

Primary Appointment:

Surgery

Secondary Appointment(s):

Physiology

Email:

[jwinkles@som.umaryland.edu](mailto:jwinkles@som.umaryland.edu)

Location:

800 West Baltimore St. Biopark Building 1, #320

Phone (Primary):

410-706-8172

Fax:

410-706-8234

[Download CV](#)

## Education and Training

- Undergraduate Degree: B.A., Biology, University of Delaware, 1977
- Graduate Degree: Ph.D., Biology, University of Virginia, 1983
- Postdoctoral Fellowship: National Institutes of Health, 1983-1986

## Highlighted Publications

Winkles, J.A. (2008). The TWEAK-Fn14 cytokine-receptor axis: Discovery, biology, and therapeutic targeting. *Nature Reviews Drug Discovery* 7:411-425.

Asrani, K., Keri R.A., Galisteo, R., Brown, S.A.N., Morgan, S.J., Ghosh, A., Tran, N.L. and Winkles, J.A. (2013). The HER2- and heregulin-1 (HRG)-inducible TNFR superfamily

member Fn14 promotes HRG-driven breast cancer cell migration, invasion and MMP9 expression. *Molecular Cancer Research* 11:393-404.

Brown, S.A.N., Cheng, E., Williams, M.S. and Winkles, J.A. (2013). TWEAK-independent Fn14 self-association and NF- $\kappa$ B activation is mediated by the C-terminal region of the Fn14 cytoplasmic domain. *PloS ONE* 8:e65248.

Cheng, E., Armstrong, C.L., Galisteo, R. and Winkles, J.A. (2013). TWEAK/Fn14 axis-targeted therapeutics: Moving basic science discoveries to the clinic. *Frontiers in Immunology* 4:473.

Zhou, H., Mohamedali, K.A., Gonzalez-Angulo, A.M., Cao, Y., Migliorini, M., Cheung, L.H., LoBello, J., Lei, X., Qi, Y., Hittelman, W.N., Winkles, J.A., Tran, N.L. and Rosenblum, M.G. (2014). Development of human serine protease-based therapeutics targeting Fn14 and identification of Fn14 as a new target overexpressed in TNBC. *Molecular Cancer Therapeutics* 13:2688-2705.

Schneider, C.S., Perez, J.G., Cheng, E., Zhang, C., Mastorakos, P., Hanes, J., Winkles, J.A., Woodworth, G.F. and Kim, A.J. (2015). Minimizing the non-specific binding of nanoparticles to the brain enables active targeting of Fn14-positive glioblastoma cells. *Biomaterials* 42:42-51.

Cheng, E., Whitsett, T.G., Tran, N.L. and Winkles, J.A. (2015). The TWEAK receptor Fn14 is an Src-inducible protein and a positive regulator of Src-driven cell invasion. *Molecular Cancer Research* 13: 575-583.

Perez, J.G., Tran, N.L., Rosenblum, M.G., Schneider, C.S., Connolly, N.P., Kim, A.J., Woodworth, G.F. and Winkles, J.A. (2016). The TWEAK receptor Fn14 is a potential cell surface portal for targeted delivery of glioblastoma therapeutics. *Oncogene* 35:2145-2155.

 [Update Your Profile](#)