

## Recent *Cell* Retractions

Chan, S.-K. and Struhl, G. (2002) *Cell* 111, 265-280

This paper presents a series of experiments that challenge the conventional view that Armadillo transduces Wingless by combining with Pangolin to form a transcriptional activator. The challenge rests principally on experiments performed by my coauthor, S.-K. Chan, the results of which are shown in the Figures 2D, 4, and 5. These experiments test the function of altered forms of Armadillo that are targeted inside or outside of the nucleus or that contain heterologous transcriptional activator or repressor domains. Recently, in seeking to extend these findings, I personally obtained the opposite result for the key negative control for the experiments in Figure 5 (Figure 5B). When confronted with this discrepancy, S.-K. Chan informed me that most of the results shown in Figures 2D, 4, and 5, including the negative control shown in Figure 5B, were either not performed or gave different results than presented in the paper. I therefore withdraw this paper and the conclusions it reports. I deeply regret and apologize for any adverse consequences that may have resulted from its publication. S.-K. Chan concurs with this retraction.

Chandok, M.R., Ytterberg, A.J., van Wijk, K.J., and Klessig, D.F. (2003). *Cell* 113, 469–482.

The above paper describes the purification and characterization of a pathogen-inducible NOS-like activity from tobacco plants and its identification as a variant form of the P subunit of the glycine decarboxylase complex. The demonstration that recombinant *Arabidopsis* variant P protein has NO-synthesizing activity was a critical piece of evidence leading to the above conclusion. Further experiments by other members of the Klessig laboratory reveal difficulties in reproducing the data with recombinant variant P and in addition suggest that the data on recombinant variant P presented in Tables 1 and 2 and in Figures 5B and 5C of this paper are unreliable. Since we cannot substantiate the major conclusion presented in this paper, we wish to retract the entire paper and its conclusions in order to avoid wasted efforts by other investigators whose studies might be influenced by the results and conclusions reported. The first author, M.R. Chandok, has not approved this retraction. We deeply regret that this serious incident occurred and sincerely apologize to our colleagues.

Yamaguchi, R. and Newport, J. (2003) *Cell* 113, 115-125

This paper (*Cell* 113, 115-125, April 4, 2003) reports results of experiments that together strongly support the conclusion that, in metazoan cells, formation of a complex consisting of the GTP binding protein Ran, the exportin Crm1, and the DNA helicase MCM plays a critical role in limiting DNA replication to a single round each cell cycle. This conclusion is largely based on two experimental results: (1) Experiments which show that a Ran mutant, RanQ69L, that binds GTP but cannot hydrolyze it inhibits incorporation of the MCM helicase into pre-replication complexes (pre-RC's) in *Xenopus* egg extracts. (2) Equally important is the observation that addition of a Ran mutant that cannot bind GTP, Ran T24N, induces both re-binding of MCM

helicase to DNA following a single round of DNA replication and induces a second round of replication. Together these results suggested that sequestration of MCM into a Crm1-Ran complex within nuclei during S phase of the cell cycle functioned to limit replication to a single round. In the course of pursuing this model further, a postdoctoral fellow in my laboratory, Dr. Peter Trabold, was able to reproduce results reported using the RanQ69L mutant. However, he was unable to reproducibly observe either the substantial reloading of MCM onto DNA or the robust re-replication reported to occur following addition of RanT24N. Occasionally, modest excess replication was observed following addition of RanT24N. However, further investigation demonstrated that this replication was most likely due to the transient permeabilization of nuclei caused by addition of RanT24N. Therefore, although experiments using RanQ69L support a model involving the Crm1-MCM complex to limit re-replication, the inability of RanT24N to induce re-replication leaves the model unproven. In light of these new results, I am withdrawing the paper and the conclusions included in it.

The first author of this article, Ryuji Yamaguchi, is not a coauthor on this retraction because he stands firmly by the data presented in the article.