Supplementary Materials

A. Parameter estimates and Assumptions adopted in the model

1). The 13 protofilaments of the microtubule are assumed to be identical; namely, the GDP-tubulin dimers form the 13_0 lattice. As a result of this simplification, the tip of the microtubule consists of all the 13 GDP-tubulin dimers from the 13 constituent protofilaments as shown in Fig. 1(B), instead of a blunt one. Therefore, the MT configuration is fully represented by one of its 13 protofilaments, $\{r_i(t), h_i(t)\}$, where i stands for the ith GDP-tubulin dimer within the protofilament, $r_i(t)$ and $h_i(t)$ are the radius and the height of ith dimer according to its upper end position, respectively (see Fig.1). The index for the GDP-tubulin dimer within the protofilament starts at the bottom as 1, and stops at the tip tubulin-dimer. In this paper, we assume the kinetochore ring as a geometric object with all the interaction is concentrated on the mass center, although it does have 5nm in thickness (1).

2). The electrostatic interaction at the nanometer length scale could be complicated by the geometries as well as the charge distributions of the charged objects. In other words, the point-charge form of the electrostatic interactions could be compromised. Therefore, to account for such deviations for the electrostatic interactions, the charge Q_{MT} and q_{kt} in our model shall be taken as the effective point charges at the mass center for the GDP-tubulin dimer and the kinetochore unit component, respectively. The electrostatic potential between the kinetochore and the microtubule is summed over all the GDP-tubulin dimers from the 13 protofilaments and the 16 identical kinetochore complex components. In the following dynamic equations, we only include the electrostatic attraction for the kinetochore ring complex, while ignoring the electrostatic interactions for the microtubule. This is because of two reasons. (a). It can be shown that, for each GDP-tubulin subunit, the electrostatic attraction from the kinetochore is negligibly small compared to the mechanical bending forces. (b). The electrostatic repulsions among the bound GDP-tubulin subunits have already been taken into account of the lateral bonds as well as the bending energies.

3). The binding energy in the model includes the electrostatic interactions, the steric repulsions as well as the specific interactions between the kinetochore ring complex and its attached GDP-tubulin subunits. This approximated harmonic potential has its minimum at the equilibrium distance between the kinetochore and the underlying dimer $R_0 - r_0 = 3.5nm$, which is deduced from the typical geometry of the kinetochore ring complex-microtubule system (1, 2). The magnitude of the binding potential is estimated from the following experiments (2, 3): since the dissociation constant for kinetochore bound with GTP-microtubule $K_d = 0.2\mu M$ (2), the binding energy between kinetochore ring complex and GTP-microtubule is $\sim k_B T \log K_d \sim 15 k_B T$. On the other hand, the kinetochore preferentially binds to GTP-microtubule as compared to the GDP-microtubule with the ratio ~ 10:1 (3), we thus can estimate that, the binding energy between the kinetochore and GDP-microtubule is $\sim k_B T \log 10 \sim 2.5 k_B T$ less than that for

the GTP-microtubule. Therefore, the binding energy between the kinetochore and the GDP- microtubule is ~ $12.5k_BT$. In simulations, the spring constant for the harmonic binding potential is chosen such that: $\frac{1}{2}k\left((4nm)^2+(3.5nm)^2\right)\sim 12.5k_BT$, where 4nm and 3.5nm correspond to maximum distance variations in the longitudinal direction and the radial direction of the binding, respectively.

4). The bare dissociation rate for the individual protofilament $k_{off}^{(0)}$ is taken as $\frac{1}{13}$ of that for the microtubule by assuming no cooperation among the protofilaments during the microtubule depolymerizations. In vivo, the concentration of free tubulin dimer in cytoplasm remains constant. The microtubule depolymerizations (polymerization) are tightly regulated by many microtubule plus-end associated proteins (4-8). The dissociation rate for kinetochore-coupled microtubule is usually about 10 times slower than the free plus-end. For simplicity, we also implicitly assume that the constriction from the kinetochore to the GDP-tubulin subunit dissociation is distinct from the regulations by other proteins in such way that: the bare off-rate is pre-determined by protein regulations, while the instantaneous effective off-rate is affected by kinetochores as $k_{off}=k_{off}^{(0)}e^{-\frac{\Delta E}{k_BT}}$, where ΔE is the potential energy stored by the tip subunits (see below). In the calculation, we choose $k_{off}^{(0)}\sim 5-10s^{-1}$ (4, 7, 8). Note: during anaphase A in cells, the dissociation of the microtubule plus-end is tightly regulated with many proteins involved (4, 7, 8). Therefore, its off-rate is always much slower than the free plus-end shrinkage in solutions.

5). The frictional drag coefficient of the configuration changes for the bound GDP-tubulin subunit is inferred from the following estimates. Consider the free plus-end microtubule depolymerization, one dissociation event includes two consecutive steps: first, the tip GDP-tubulin subunit needs to fully curl out according to its preferred angle; secondly, this dimer falls off at this configuration. Here, we assume the second step is instantaneous. Therefore, in the mean-field approximation, the bare dissociation rate of protofilaments $k_{off}^{(0)}$ determines the time ($\sim\frac{1}{k_{off}^{(0)}}$) for the bound GDP-tubulin subunits to fully bend out for the first time. The dynamic equation for the position changes of the free-end bound GDP-tubulin subunits is: $\gamma_{MT}\frac{dr_i}{dt}=-\frac{\partial V_{free}}{\partial r_i}$, where the potential for bound GDP-tubulin subunits without the kinetochore influence V_{free} is

$$V_{free}\left(\left\{\vec{r}_i^{(n)}(t)\right\}\right)=\sum_{i=1}^{N}\left(\overbrace{\frac{1}{2}\kappa\left(\varphi_i-\varphi_{i-1}-\varphi^{(0)}\right)^2}^{\substack{bending\\ energy}}+\overbrace{A\left(\Delta r_i\right)\left(\frac{\Delta r_i}{\Delta r^{(0)}}\right)^2 e^{-\frac{\Delta r_i}{\Delta r^{(0)}}}}^{\substack{lateral\\ bond}}\right).$$

Starting from the up-straight configuration as the initial condition, we integrate this equation over time. We then equal the time for the tip subunit to fully curl out for the first time to $\frac{1}{k_{off}^{(0)}}$. Thus, in this way, we numerically determine the average frictional drag coefficient for the configuration changes of the bound GDP-tubulin subunit: $\gamma_{MT} \sim 10-20\frac{\mathrm{pN}\cdot\sec}{\mu m}$ for $k_{off}^{(0)} \sim 5-10 s^{-1}$ (4, 7, 8). Meanwhile, the frictional drag coefficient for the kinetochore is $\gamma_{kt} \sim 5-10\frac{\mathrm{pN}\cdot\sec}{\mu m}$ (9-11) if it is connected by the full chromatid, whereas it will be much smaller in the system that the chromatid arms are being severed (12). For the in vitro experiments (13), the frictional drag coefficient for Dam1 kinetochore ring could be estimated by the Einstein relationship $D_{diff} = \frac{k_B T}{\gamma_{kt}}$. Therefore, in this case, $D_{diff} = 5.4\times10^{-10} cm^2/s$ leads to $\gamma_{kt} = 0.08\frac{pN.\sec}{\mu m}$. The kinetochore thus moves at a relatively faster time scale than the underlying GDP-tubulin subunits.

6). There could be 1~50 kinetochore microtubules (kMTs) per chromatid depending on cell types (14). In our model, we only consider the situation that one kMT per chromatid. If we assume that the different kMTs in the same chromatid become quickly synchronized such that the individual kinetochore poleward translocation is at the same velocity, then the stalling force will be simply the stalling force from one microtubule multiplied by the number of the kMTs in the same chromatid.

B. Full dynamic equations

The dynamic equation for the kinetochore is:

$$\gamma_{kt}\frac{dH}{dt} = -\frac{\partial V}{\partial H}$$

$$= -\left(\sum_{n=1}^{13}\sum_{m=1}^{16}\sum_{i=1}^{tip-1}\frac{q_{kt}Q_{MT}}{4\pi\varepsilon\varepsilon_0}\frac{e^{-\frac{l_{n,m,i}}{\lambda_D}}}{l_{n,m,i}^2}\left(\frac{1}{l_{n,m,i}}+\frac{1}{\lambda_D}\right)\left(H(t)-\frac{1}{2}\left(h_i(t)+h_{i-1}(t)\right)\right)\right)$$

$$-\left(k\left(H(t)-\frac{1}{2}\left(h_{tip}(t)+h_{tip-1}(t)\right)\right)-13\kappa\left(\frac{\left(\varphi_t-\varphi_{t-1}-\varphi^{(0)}\right)}{\sqrt{l^2-\left(r_t-r_{t-1}\right)^2}}\right)\frac{\sin\varphi_{tip}}{\cos\varphi_{tip}}\right)+\xi_{kt}$$

$l_{n,m,i}$ is the distance between the *i*th GDP-tubulin subunit of the *n*th protofilament and the *m*th kinetochore component.

$$l_{n,m,i} = \sqrt{R_0^2+\frac{1}{4}\left(r_i(t)+r_{i-1}(t)\right)^2-\frac{1}{2}R_0\left(r_i(t)+r_{i-1}(t)\right)\cos\left(\frac{2\pi n}{13}-\frac{2\pi m}{16}+\theta_0\right)+\left(H(t)-\frac{1}{2}\left(h_i(t)+h_{i-1}(t)\right)\right)^2}$$

$\frac{1}{2}(r_i + r_{i-1})$ and $\frac{1}{2}(h_i + h_{i-1})$ are the radius, the height of the mass center of the *i*th tubulin-subunit, respectively. The first term comes from the electrostatic interactions; the second term originates from the harmonic binding potential; the third term stems from the local bound tubulin subunit bending strength. The thermal fluctuation ξ_{kt} follows the Einstein relationship $\langle \xi_{kt}(t_1, \vec{r}_1) \xi_{kt}(t_2, \vec{r}_2) \rangle \sim \frac{\gamma_{kt}^2 D_{kt}}{dt} \delta(t_1 - t_2) \delta(\vec{r}_1 - \vec{r}_2)$. The dynamic equations for the tip subunit and the next to the tip read:

$$\gamma_{MT} \frac{dr_t}{dt} = -\frac{\partial V}{\partial r_t}$$

$$= -\left(\kappa \left(\frac{(\varphi_t - \varphi_{t-1} - \varphi^{(0)})}{\sqrt{l^2 - (r_t - r_{t-1})^2}} \right) + f_{bond} + f_{binding} \right)$$

, where *t*=tip, or tip-1. f_{bond} is the force originated from the lateral bond.

If $\Delta r_i \le 2\Delta r^{(0)}$, $f_{bond} = A e^{-\frac{\Delta r_i}{\Delta r^{(0)}}} \left(\frac{\Delta r_i}{\Delta r^{(0)}} \right) \left(\frac{1}{\Delta r^{(0)}} \right) \left(-\frac{\Delta r_i}{\Delta r^{(0)}} + 2 \right) \left(2 \sin \frac{\pi}{13} \right)$; otherwise, the lateral bond breaks, no force is invoked, therefore $f_{bond} = 0$. The lateral bond length between the neighboring tubulin subunits in the adjacent protofilaments is: $\Delta r_i = 2(r_i - r_0) \sin \frac{\pi}{13} + \Delta r^{(0)}$. $f_{binding}$ is the force from the binding potential. For the tip GDP-tubulin subunit,

$$f_{binding} = \frac{k}{2} \left(\left(\frac{r_{tip}(t) + r_{tip-1}(t)}{2} - r_0 \right) - \left(\frac{h_{tip}(t) + h_{tip-1}(t)}{2} - H(t) \right) \frac{\sin \varphi_{tip}}{\cos \varphi_{tip}} \right);$$

for the next (tip-1) subunit,

$$f_{binding} = \frac{k}{2} \left(\left(\frac{r_{tip}(t) + r_{tip-1}(t)}{2} - r_0 \right) - \left(\frac{h_{tip}(t) + h_{tip-1}(t)}{2} - H(t) \right) \frac{\sin \varphi_{tip-1}}{\cos \varphi_{tip-1}} \right)$$

The dynamic equations for the rest of the subunits are:

$$\gamma_{MT} \frac{dr_i}{dt} = -\frac{\partial V}{\partial r_i}$$

$$= -\left(\kappa \left(\frac{(\varphi_i - \varphi_{i-1} - \varphi^{(0)})}{\sqrt{l^2 - (r_i - r_{i-1})^2}} - \frac{(\varphi_{i+1} - \varphi_i - \varphi^{(0)})}{\sqrt{l^2 - (r_i - r_{i-1})^2}} - \frac{(\varphi_{i+1} - \varphi_i - \varphi^{(0)})}{\sqrt{l^2 - (r_{i+1} - r_i)^2}} \right) + f_{bond} \right)$$

C. Boundary conditions

For the tip GDP-tubulin dimer, the boundary condition is open. For the bottom GDP-tubulin subunit, the position is fixed as the radius $r_1 = 12.5nm$, the height $h_1 = 0nm$ and the orientation of the bottom subunit $\varphi_1 = 0$ throughout the simulations. The kinetochore complex ring is rigid, which is realized by fixing the radius of the kinetochore as 16nm in

the simulations. The kinetochore is also not penetrable. In simulation, this constraint is enforced by the hard wall repulsion, which becomes effective if the distance between the kinetochore and the bound GDP-tubulin subunit is less than 0.2nm. We can show that the qualitative features of our results do not change much if the length cut-off varies.

D. Initial conditions

The protofilament of the GDP-tubulin subunits is in the up-straight configuration $\varphi_i = 0$ for $i = 1,2,...N$ and the kinetochore is at same height of the mass center of the tip GDP-tubulin subunit.

E. Simulation details

In our simulations, there are 500 GDP-tubulin subunits in one protofilament (the microtubule is initially thus $4\mu m$ long). The positions of each GDP-tubulin subunit and the kinetochore ring complex are calculated by integrating the corresponding dynamic equations over the time step dt. We choose $dt = 5\times10^{-5}$ sec to maintain good convergence (to test the convergence of the resulting dynamic trajectories, we also use $dt = 5\times10^{-6} \sim 5\times10^{-8}$ sec, and the results are essentially identical to the case of $dt = 5\times10^{-5}$ sec). Because we focus on the deterministic forces for kinetochore translocation, we shut off the thermal noise throughout our simulations. In the simulation, the potential energy is scaled in k_BT ($1k_BT \sim 4\,pN\cdot nm$), the length is scaled in *nm*,

At each simulation time step, we calculate the dissociating rate for the tip GDP-tubulin subunit $k_{off} = k_{off}^{(0)} e^{-\frac{\Delta E}{k_BT}}$, where

$$\Delta E = \frac{1}{2}\kappa\left(\varphi_{tip} - \varphi_{next} - \varphi^{(0)}\right)^2 + \frac{k}{2}\left(\left(r_0 - \frac{1}{2}\left(r_{tip}(t) + r_{next}(t)\right)\right)^2 + \left(H(t) - \frac{1}{2}\left(h_{tip}(t) + h_{next}(t)\right)\right)^2\right)$$
$$+ A\left(\Delta r_{tip}\right)\left(\frac{\Delta r_{tip}}{\Delta r^{(0)}}\right)^2 e^{-\frac{\Delta r_{tip}}{\Delta r^{(0)}}}$$

At each simulation step, we also obtain the potential landscape for the kinetochore $V_{kt}(H(t))$ as the function of the kinetochore position $H(t)$ while keeping the GDP-tubulin subunit positions fixed.

$$V\left(\vec{R}_m(t), \left\{\vec{r}_i^{(n)}(t)\right\}\right) = \overbrace{\sum_{n=1}^{13}\sum_{m=1}^{16}\sum_{\substack{i=1\\(i\neq K)}}^{N} \frac{q_{kt}Q_{MT}e^{-\frac{\left|\vec{R}_m - \frac{1}{2}\left(\vec{r}_i^{(n)} + \vec{r}_{i-1}^{(n)}\right)\right|}{\lambda_D}}}{4\pi\varepsilon\varepsilon_0 \left|\vec{R}_m - \frac{1}{2}\left(\vec{r}_i^{(n)} + \vec{r}_{i-1}^{(n)}\right)\right|}}^{\text{electrostatic attractions}} + \underbrace{\frac{k}{2}\left(\left(\vec{R} - \frac{1}{2}\left(\vec{r}_K(t) + \vec{r}_{K-1}(t)\right)\right) - \left(R_0 - r_0\right)\right)^2}_{\text{binding energy}}$$

We keep tracks of the lifetime t_{life} of the GDP-tubulin subunit as the tip. At mean field level approximation, if $t_{life} > \frac{1}{k_{off}}$, then the tip will dissociate. Accordingly, the next GDP-tubulin subunit becomes the tip, and the simulation continues. If the tip GDP-tubulin subunit dissociates before the kinetochore gets to the next subunit $H(t) > h_{next}(t)$, then the kinetochore will fall off together with the tip GDP-tubulin subunit, and the simulation stops.

Reference

1. Miranda, J. J. L., Wulf, P. D., Sorger P. K. & Harrison, S. C. (2005) *Nat. Struct. Mol. Biol.* **12**, 138-143.
2. Westermann, S., Avila-Sakar, A., Wang, H. W., Niederstrasser, H., Drubin, D. G., Nogales, E. & Barnes, G. (2005) *Mol. Cell* **17**, 277-290.
3. Severin, F. F., Sorger, P. K. & Hyman, A. A. (1997) *Nature* **388**, 888-891.
4. Desai, A., Verma, S., Mitchison, T. J. & Walczak, C. E. (1999) *Cell* **96**, 69-78.
5. Niederstrasser, H., Salehi-Had, H., Gan, E. C., Walczak, C. & Nogales, E. (2002) *J. Mol. Biol.* **316**, 817-828.
6. Roger, G. C., Roger, S. L., Schwimmer, T. A., Ems-McClung, S. C., Walczak, C. E., Vale, R. D., Scholey, J. M. & Sharp, D. J. (2004) *Nature* **427**, 364-370.
7. Walker, R. A., O'Brien, E. T., Pryer, N. K., Soboeiro, M. F., Voter, W. A., Erickson, H. P. & Salmon, E. D. (1988) *J. Cell Biol.* **107**, 1437-1448.
8. Helenius, J., Brouhard, G., Kalaidzidis, Y., Diez, S. & Howard, J. (2006) *Nature* **441**, 115-119.
9. Alexander, S. P. & Reider, C. L. (1991) *J. Cell Biol.* **113**, 805-815.
10. Nicklas, R. B. (1983) *J. Cell Biol.* **97**, 542-548.
11. Marshall, W. F., Marko, J. F., Agard, D. A. & Sedat, J. W. (2001) *Curr. Biol.* **11**, 569-578.
12. Skibbens, R. V., Skeen, V. P. & Salmon, E. D. (1993) *J. Cell Biol.* **122**, 859-875.
13. Westermann, S., Wang, H. W., Avila-Sakar, A., Drubin, D. G., Nogales, E. & Barnes, G. (2006) *Nature* **440**, 565-569.
14. Maiato, H., Deluca, J., Salmon, E. D. & Earnshaw, W. C. (2004) *J. Cell Sci.* **117**, 5461-5477.