The nucleus is a cellular compartment that hosts several macro-molecular machines displaying a highly complex spatial organization. This tight architectural orchestration determines not only DNA replication and repair but also regulates gene expression. In budding yeast microtubules play a key role in structuring the nucleus since they condition the Rabl arrangement in G1 and chromosome partitioning during mitosis through their attachment to centromeres via the kinetochore proteins. Recently, we have shown that upon quiescence entry, intranuclear microtubules emanating from the spindle pole body elongate to form a highly stable bundle that spans the entire nucleus. Here, we examine some molecular mechanisms that may underlie the formation of this structure. As the intranuclear microtubule bundle causes a profound re-organization of the yeast nucleus and is required for cell survival during quiescence, we discuss the possibility that the assembly of such a structure participates in quiescence establishment.

**Introduction**

Cells perpetually face the decision to proliferate or to enter a non-dividing state. Non-dividing states may be irreversible, such as senescence, or only temporary. Quiescence, a state defined as a reversible arrest of proliferation, is probably the most common cellular situation found on earth, as it concerns a wide range of cells, from microbes to human stem cells. Quiescent cells are confronted with quite a few challenges. On the one hand, quiescent cells need to preserve their ability to proliferate, sometimes over several years. As they age, quiescent cells must cope with extrinsic or intrinsic harmful events that cause the accumulation of damaged macromolecules. The inability to handle these stresses can ultimately lead to cell death. Additionally, for the sake of tissues, organisms, and the species itself, quiescent cells must produce offspring born “damage-free.” On the other hand, cells must enter quiescence and return into the proliferation cycle in a tightly regulated manner, uncontrolled transitions being potentially deleterious for the whole organism, as exemplified by stem cell depletion or cancer. Finally, in the case of microorganisms competing for their environmental niche, quiescence exit must be swift to ensure the prevalence of the species. Therefore, quiescence is at the heart of crucial biological issues including development, aging, and evolution.

Quiescence encompasses highly diverse physiological situations that differ not only depending on the cell type but also, in the case of the same type of cells, depending on both the stimuli that triggered quiescence entry (contact inhibition, exhaustion of growth factors or nutrients, hypoxia, or other) and on the time spent in quiescence. In single cell eukaryotes like *S. cerevisiae*, quiescence is primarily induced by the limitation of essential nutrients. When the carbon source is exhausted, most cells—but not all—enter quiescence from the G1 phase of the cell cycle. Quiescent yeast cells are not simply resting G1 cells as they acquire a variety of specific
properties, including a gene expression profile that differs significantly from the one displayed by G1 arrested cells. In addition, the establishment of quiescence in yeast is accompanied by a decrease in protein synthesis rate, the accumulation of storage molecules, and the reorganization of various cellular machinery. Indeed, upon quiescence entry, the mitochondria tubular network fragments, the proteasome exits the nucleus and joins cytoplasmic structures called proteasome storage granules, actin filaments collapse into actin bodies, and various chaperones and metabolic enzymes are re-localized. Some of these reorganizations have been described in both S. cerevisiae and S. pombe, but also in metazoans, suggesting that they may be conserved among eukaryotes. The assembly of quiescence specific structures implies a cell’s commitment to this specific cellular state. As some of these structures have been shown to be required for survival and/or exit from quiescence, one key question is to now decipher whether the observed reorganizations are a passive consequence of the cell’s entry into quiescence or if they actively participate in the process of quiescence establishment.

The Nucleus is Drastically Reorganized upon Quiescence Establishment

In the G1 phase of the cell cycle, the budding yeast nucleus is arranged in a Rabl configuration. Each centromere is attached via its kinetochore complex to a short nuclear microtubule (<300 nm) that emanates from the spindle pole body (SPB, the yeast equivalent of the centrosome). While centromeres are highly clustered next to the SPB, most of the telomeres are localized into 8–10 foci distributed over the nuclear periphery. The nucleolus, built around the rDNA, is opposed to the SPB on the other half of the nucleus. This typical organization strictly depends on nuclear microtubules. Indeed, it was shown in vivo that microtubule depolymerization causes centromere declustering and in silico modeling predicts that, in the absence of microtubules, the rDNA would fail to position opposite to the SPB. Besides, telomere cluster formation and distribution at the nuclear periphery was shown to be influenced by the amount of the silencing protein Sir3. Therefore, the nuclear architecture is highly intricate and its compartmentalization tightly controlled.

Upon quiescence entry, we have shown that the S. cerevisiae nucleus is drastically reorganized. SPB-associated nuclear microtubules elongate to form an extended and extremely stable bundle that spans the entire nucleus. Because centromeres remain attached to the microtubule ends, they are mostly found at the tip of the microtubule bundle, opposite to the SPB. Yet, as microtubules within the bundle are not all of the same length, centromeres can occasionally be found close to the SPB or along the length of the microtubule array. The nucleolus is displaced to one side of the microtubule array, often close to the SPB (Fig. 2D). Of note, the size of the nucleolus is greatly diminished (Fig. 2B), in agreement with the drastic decrease in ribosome synthesis and translation rate in quiescent cells. In mutants that do not assemble a microtubule array in quiescence, the centromeres and the nucleolus are no longer displaced, demonstrating that microtubule elongation and stabilization in quiescence are causing the nuclear rearrangements. We have also found that the silencing proteins Sir2 and Sir3 are localized into 2 to 3 foci at the nuclear periphery of quiescent cells, probably reflecting an hyper-clustering of telomeres (D.L. and I.S., unpublished results; Fig. 2E and F). Interestingly, these foci are often localized next to the SPB (D.L. and I.S., unpublished results; Fig. 2F). The existence of a possible interplay between microtubules and telomere rearrangement remain to be investigated. Yet, it is worth noting that in early prophase of meiosis I, the yeast nucleus adopts an organization called the “bouquet” in which, like in quiescent cells, the telomeres gather together close to the SPB. Does the telomere congregation next to the SPB observed in quiescence and meiosis involve similar molecular processes? In the bouquet arrangement, centromeres are no longer found in proximity to the SPB like they are in G1, but rather are scattered throughout the nucleoplasm. Intriguingly, upon transition from the Rabl configuration to the bouquet arrangement, the assembly of a short-lived intranuclear microtubule bundle was reported. Concomitantly, centromeres are found transiently arranged along “a line,” but whether the lining up of the centromeres takes place along the microtubule bundle has not been investigated. Ultimately, the microtubule structure disappears, and, at the pachytene stage, centromeres are unleashed into the nucleoplasm. One interesting possibility is that the microtubule bundle observed in meiosis may be comparable to the structure assembled in quiescent cells. Another intriguing analogy between quiescence and meiosis is that both processes are induced by nutrient starvation. Are the signals and the mechanisms responsible for microtubule array formation similar
for both cellular situations? The discovery of molecular congruence between these two nuclear reorganizations would be striking.

**Molecular Mechanisms Underlying Microtubule Array Formation in Quiescent Cells**

Microtubules are composed of α- and β-tubulin heterodimers that are assembled after a nucleation step typically requiring the gamma tubulin complex. In proliferating yeast cells, the gamma tubulin complex is embedded into the SPB, and while the microtubule “minus end” stays closely associated with this structure, the opposite end, the “plus end,” is highly dynamic and alternates between periods of growth, pause, and shrinkage. In yeast, like in other eukaryotes, microtubule dynamic is influenced by a plethora of microtubule-associated proteins (MAPs) that can either stabilize or destabilize microtubules.38-41

Quiescent cells assemble an array of microtubules that spans the entire nucleus, sometimes even causing the deformation of the nuclear membrane.35 The net elongation of microtubules could result from an increased polymerization rate, a decreased depolymerization rate, a diminished catastrophe frequency, an enhanced rescue frequency, or a combination of all these events. Upon quiescence entry microtubules may grow from the SPB in various directions until they reach the nuclear membrane. Due to the spatial constraint imposed by this physical barrier, growing microtubules would slide along the membrane toward the opposite side of the nucleus, where they would reach their “maximum” length (Fig. 3A). Alternatively,
packing forces due to microtubule bundling proteins or to interaction between neighboring kinetochores may result in multiple microtubules simultaneously elongating in front of the SPB in a constrained manner (Fig. 3B).

In quiescence nuclear microtubules are not only long but amazingly stable. This stability could be actively triggered by enhanced microtubule stabilization activity and/or may be the consequence of the depletion of microtubule destabilizing proteins. We have found that a large number of MAPs that influence microtubule dynamics are not required for the formation and the stability of microtubule array, even though some of these MAPs were clearly detected along the microtubule bundle.55 This is the case for Bim1, the EB1 homolog known to decrease the catastrophe frequency.52 Astonishingly, we have found that dynein, a microtubule minus-end-directed motor and its regulator, the dynactin complex, are required for the microtubule array formation. Yet, in quiescent cells, none of these proteins could be detected in the nucleus but were rather localized on the cytoplasmic face of the SPB.35 If these MAPs are indeed absent in the nucleus, how could they affect the formation of a nuclear microtubule structure? In budding yeast, the SPB traverses the nuclear envelope.40 Thus, one possibility could be that deletion of cytoplasmic proteins, such as dynein, alters the composition and/or architecture of the SPB, which causes its incapacity to nucleate (or stabilize) microtubules inside the nucleus. In S. pombe it was suggested that dynein deletion may impede on SPB integrity.43,44 Whether the SPB is affected in dynein and dynactin mutants in quiescent S. cerevisiae is not known.

At the other end of the bundle, the plus ends of microtubules may be “capped” by proteins, thereby preventing their depolymerization. One obvious candidate is the kinetochore complex. In proliferating cells it was shown that microtubules that are attached to kinetochore are more stable than unattached ones.55-57 In quiescence the microtubule-kinetochore attachment could be locked, leading to an enhanced kinetochore microtubule-stabilizing activity and the “freezing” of microtubule plus end. A peculiar observation is the localization of members of the Dam complex, a yeast kinetochore sub-module, all along the microtubule bundle. In vitro, the Dam complex forms a ring around the microtubule.58-61 Could the Dam complex, by forming collars all along the microtubules participate in microtubule stabilization and/or bundling in quiescent cells? Deciphering the roles of each kinetochore sub complex will likely shed light on the molecular mechanisms driving the formation of the atypically stable bundle of parallel microtubules assembled in quiescent yeast cells.

The Nuclear Microtubule Bundle May Influence Gene Expression and Thereby be Required for Quiescence Establishment

Whatever the molecular mechanisms involved in the formation of the microtubule bundle in the nucleus of quiescent cells, one key question is: what is the physiological “raison d’être” of this structure? Does it play a crucial role in quiescence establishment? Is this structure needed for the fitness during quiescence exit? We have found that mutants unable to assemble a microtubule bundle in quiescence are impaired for quiescence survival.35 One intriguing possibility could be that the chromosome rearrangement imposed by the formation of the microtubule array contributes to the specific gene expression profile adopted by the cells upon quiescence entry. Indeed, although chromosomes display confined movement in interphase,52 it was proposed that the localization of a gene within the nucleus correlates with its transcriptional activity. In the current model, genes expressed at a basal level preferentially localize toward the center of the nucleus while actively silenced chromosome regions are associated with the nuclear membrane. Massively transcribed genes would be located rather close to nuclear pores, a proximity that is supposed to improve the export efficiency of the newly synthesized mRNA (for a review see ref. 30 and refs. herein). By inducing the displacement of most centromeres and the relocalization of the nucleolus, the microtubule array would cause severe variation in gene position within the nucleolus, the microtubule array would cause severe variation in gene position within the nuclear compartment and thereby modify the transcriptional profile of the cell. Therefore, by modifying gene expression, the nuclear microtubule array may be needed for quiescence establishment. Mutants that are not able to assemble this structure would fail in modifying their transcriptome and, consequently, would be incapable of installing quiescence. This would explain why such mutants have a reduced viability in this non-proliferating cellular state. Interestingly, drastic and reversible chromosome displacements have been reported upon proliferation and/or quiescence transitions in primary...
human fibroblasts.\textsuperscript{53} Like in yeast, the nuclear micro-environment influences gene regulation in mammals.\textsuperscript{54} Therefore, chromosomal re-localization may also be involved in quiescence establishment in metazoan cells.

**Microtubule and/or Centromere Attachment and the Fitness of Quiescence Exit**

When yeast cells proliferate, centromeres stay connected to the microtubule plus end throughout cell cycle progression. In fact, the centromere-microtubule attachment is lost for only a few minutes, which is just the time needed to replicate centromeric DNA.\textsuperscript{55-57} In mitosis, a single unattached centromere can activate the spindle assembly checkpoint and delay cell division until the defect is solved.\textsuperscript{58,59} In quiescent cells centromeres are mostly localized at the tip of the microtubule bundle. When quiescence exit is triggered by nutrient addition, the nuclear microtubule array slowly shortens, and about an hour after re-feeding, cells have recovered a typical Rabl configuration. During this entire process, microtubules remain bundled and the centromeres stay apparently attached to the depolymerizing microtubule plus ends. In fact, yeast cells appear to keep their centromeres attached to the microtubule plus end throughout the quiescence cycle, just as they do during proliferation.\textsuperscript{39} Keeping centromeres attached to microtubules during quiescence could be a mechanism to avoid the recapture step that would delay re-entry into the cell cycle. Indeed, for single cell eukaryotes like yeast that are in direct competition for nutrients with other species, quiescent exit must be fast since it conditions the survival of the species in the environmental niche.

Most cells unable to assemble a microtubule bundle die after a few days in quiescence. Interestingly, the survivors show a delay in resuming the cell cycle upon refeeding and not many are capable of producing healthy progeny. In fact, most of the cells either do not divide or give rise to a micro-colony.\textsuperscript{35} Mutants unable to assemble a microtubule bundle may experience genome instability in quiescence or chromosome segregation defects upon quiescence exit, processes that would ultimately lead to unfit progeny.

Upon quiescence establishment, the formation of a stable microtubule bundle inside the nucleus may be a \textit{S. cerevisiae} specificity that is linked to the particular architecture of its spindle pole body. Indeed, the budding yeast SPB is permanently inserted across the nuclear membrane and displays a cytoplasmic and a nuclear face that are both capable of nucleating microtubule. Intriguingly, in mammals, quiescence entry has been associated with the formation of a primary cilium, a microtubule-containing structure that is nucleated from the older centriole of the centrosome.\textsuperscript{60} It has been proposed that the primary cilium may function as a "physical checkpoint" for cell cycle re-entry since its resorption is apparently required for quiescence exit.\textsuperscript{61} Might the microtubule intranuclear bundle assembled in \textit{S. cerevisiae} be the distant cousin of the primary cilium?

**Disclosure of Potential Conflicts of Interest**

No potential conflict of interest was disclosed.

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