The retinoblastoma family of proteins are key cell cycle regulatory molecules important for the differentiation of various mammalian cell types. The retinoblastoma protein regulates transcription of a variety of genes either by blocking the activation domain of various activators or by active repression via recruitment to appropriate promoters. We show here that the retinoblastoma family of proteins functions as direct transcriptional repressors in a heterologous yeast system when fused to the DNA binding domain of Gal4. Mapping experiments indicate that either the A or the B domain of the pocket region is sufficient for repression in vivo. As is the case in mammalian cells, a phosphorylation site mutant of the retinoblastoma protein is a stronger transcriptional repressor than the wild type protein. We show that transcriptional repression by pRb is dependent on CLN3 in vivo. Furthermore, the yeast histone deacetylase components, RPD3 and SIN3, are required for transcriptional repression.

The retinoblastoma protein (pRb) is a key regulatory molecule important for cell cycle control and differentiation in a number of mammalian cell types (1). p107 and p130 are two related family members, who share both sequence and functional properties with the retinoblastoma protein (2). The three proteins show greatest similarity within the so-called pocket domain, consisting of A and B subdomains. The A and B subdomains have been reported to show weak similarity to the pocket domain, consisting of A and B subdomains. The A and B subdomains have been reported to show weak similarity to the pocket domain, consisting of A and B subdomains. The A and B subdomains have been reported to show weak similarity to the pocket domain, consisting of A and B subdomains.

The retinoblastoma protein regulates gene expression presumably through its interaction with transcriptional activators such as E2F1 (8–10), Myod (11), Elf1 (12), c-Myc (13), PU.1 (4), ATF2 (14), and UBF (15). E2F1-responsive genes are repressed by pRb via the interaction of pRb with the activation domain of E2F1, and this interaction is a phosphorylation-dependent event. Phosphorylation of pRb inhibits its ability to bind the activation domain of E2F1 and the subsequent activation of E2F1-dependent transcription is sufficient for cells to proceed through cell division (16, 17). Previous studies have shown that the retinoblastoma protein can be ectopically expressed in Saccharomyces cerevisiae and that cyclin-dependent phosphorylation of the protein in yeast mimics that observed in mammalian cells (18).

In mammalian cells, the retinoblastoma protein bears intrinsic transcriptional repression properties when tethered to DNA via the heterologous Gal4 DNA binding domain, and this function is dependent on an intact pocket region (19–22). The retinoblastoma protein has been shown to interact with several proteins that may be relevant to its ability to directly inhibit transcription in vivo. pRb has been shown to interact with histone deacetylase (HDAC) and presumably inhibits transcription by virtue of deacetylating chromatin in the vicinity of the transcription initiation site (23–25). Chromatin effects may also be mediated by the interaction of pRb with hSWI/SNF (26–28). Another potential mechanism of transcriptional repression by pRb is via its interaction with TAFII250 (29). TAFII250 possesses an intrinsic kinase activity that is inhibited upon interaction with the retinoblastoma protein (30). In vitro studies have shown that Gal4-pRb actively represses transcription on chromatinized templates but not naked DNA templates suggesting a biochemically distinct mechanism of repression from that of E2F inhibition (31).

Since many aspects of transcriptional control are conserved from yeast to man, we examined the transcriptional properties of the retinoblastoma family in the yeast S. cerevisiae. Using fusions of pRb, p107, and p130 to the heterologous Gal4 DNA binding domain we studied the intrinsic transcriptional repression properties of these molecules in vivo. Our results show that the retinoblastoma family of proteins can function as direct transcriptional repressors in yeast with properties similar but distinct from those observed in mammalian cells.

EXPERIMENTAL PROCEDURES

Strains and Growth Conditions—Yeast S. cerevisiae strain GGY1::GG9 is a derivative of GGY1 (gal4, gal80, tyr1, ade, leu2, his3, ura3) harboring a lacZ reporter construct integrated at the UR3 locus (32). We used a marker swap protocol (33) to generate the strain GGY1::GG9 in which the TRP1 gene has been eliminated by insertion of the HIS3 gene. Strains NLY2::SS38–3 and NLY2::SS36 are derivatives of NLY2 (gal4, gal80, ura3, his3, leu2, trpl, lyp2) harboring a lacZ reporter construct from the plasmids SS38–3 and SS36, respectively, integrated at the UR3 locus (34). S. cerevisiae strains FT5Δsin3 and FT5Δpdp3 (35) are derivatives of FT5 (ura3, trpl, his3, leu2) carrying Δsin3::HIS3 and Δpdp3::HIS3 alleles and were kindly provided by Dr. K. Struhl. Δcin1, Δcin2, and Δcin3 deletions were constructed in strain W3031B (ade2, trpl, leu2, his3, ura3) by a PCR-based gene deletion method using the pFA6a-HisMX6 vector as described in (36). For monitoring the activity of LexA fusions, plasmid JK1621 (2 μ, URA3) containing the CYC1-lacZ reporter was used (37). Plasmid-containing yeast strains were grown at 30 °C in 5 ml of selective synthetic drop-out media (38), with glucose as a carbon source. Yeast cultures were grown overnight to an optical density at 600 nm (A600) between 1–1.5. Escherichia coli strain DH5α was used for the construction and propagation of plasmid vectors.
Plasmid Constructs and Yeast Transformation—Gal4 (1–147 aa) fusion constructs were constructed using the pAS2 vector (CLONTECH). pAS2 containing Gal4-p130 and Gal4-p107 (39) were kindly provided by Dr. A. Yee. Plasmid vectors (pECE) containing mouse pRb or the phosphorylation mutant Δp34 (40) were obtained from Dr. Eldad Zacksenhaus. Gal4-pRB and Gal4-Δp34 fusions were constructed by ligation of a NcoI + BamHI fragment isolated from the parental pECE vectors into NcoI + BamHI-cut pAS2. Gal4-Δ593–921 was constructed by deleting the PstI fragment from Gal4-pRB in pAS2. A fragment containing pRB aa 1–293 was generated by digesting pECE-pRB with PvuII, filling in the ends with Klenow and then digesting with NcoI. This 0.7-kb fragment was then ligated into pAS2 cut with NcoI + SalI generating Gal4-Δ234–921. A fragment containing only aa 593–921 (B-domain and C-terminus) was cut with PstI from Gal4-pRB in pAS2, and ligated into PstI-digested pAS2. Deletions of human pRB contained in the vector pSK+ were obtained from Dr. Ed Harlow (41). These deletions were isolated as EcoRV + BamHI fragments and then ligated into SmaI + SalI generating Gal4-Δ180,303–421, Gal4-Δ180,622–714, Gal4-Δ180,773–909, respectively. Human deletion Gal4-Δ180,603–909 was constructed by deleting the PstI fragment from Gal4-Δ180,603–909 in pAS2.

pLexA fusion constructs were constructed into the pBTM116 vector (2 μg, TRP1). The LexA-pRB fusion was constructed by ligation of an EcoRI + SalI fragment containing pRB (from the Gal4-pRB construct) into EcoRI + SalI-cut pBTM116. Plasmid Gal4-p150 was digested with NheI, and the ends were filled in with Klenow and then digested with PstI. The fragment containing the p130 sequence was ligated into pBTM116 cut with SmaI + PstI, generating the LexA-p130 fusion. Yeast cells were transformed by the lithium acetate method (42).

β-Galactosidase Assays—Specific β-galactosidase activities were determined from yeast cultures by the method described in Ref. 38. Each assay was performed in triplicate from at least three independent transformants. The standard error was <15% except for reporter SS36, for which the error was under 20%.

Protein Extracts and Western Blotting—Protein extracts were prepared as described previously (38) except that the breaking buffer contained 1 mM NaCl. Western blots were performed as described previously (43). 100 μg of protein from each extract preparation were analyzed except for constructs Gal4-Δ593–921, Gal4-Δ180,603–909 and Gal4-Δ1–592 for which we used 300, 400, and 500 μg of proteins, respectively. Proteins were resolved on a 12.5% SDS-polyacrylamide gel and transferred to a nitrocellulose membrane. The nitrocellulose membrane was incubated with a 1:400 dilution of Gal4 DNA binding domain monoclonal antibody (Santa Cruz Biotechnology), followed by a 1:3000 dilution of goat anti-mouse peroxidase-conjugated secondary antibody (Bio-Rad). Signals were detected by enhanced chemiluminescence as per the manufacturer’s protocol (Pierce).

RESULTS

We sought to determine whether the retinoblastoma family of proteins could function as direct transcriptional repressors in a heterologous yeast system using fusion proteins of pRB, p107, and p130, respectively, to the heterologous Gal4 DNA binding domain (Fig. 1). We tested their transcription function in vivo using chromosomally integrated lacZ reporter constructs containing the yeast GAL1 core promoter having Gal4 binding sites either upstream or downstream of Gcn4 binding sites (Fig. 2, A and B). The data presented in Fig. 2, A and B show that all three constructs inhibited transcription of the lacZ reporter construct by ∼50% regardless of whether the Gal4 binding sites were upstream or downstream of the Gcn4 binding sites. In addition, this repression was dependent on binding to the Gal4 sites as a reporter construct lacking Gal4 binding sites was unaffected by Gal4-pRB, Gal4-p107, or Gal4-p130 (Fig. 2C). These results mimicked those using similar fusion proteins in mammalian cells (19–21).

To identify the regions of pRB necessary for transcriptional repression in yeast, we constructed in frame fusions to the Gal4 DNA binding domain of several deletion mutants spanning both the N-terminal and C-terminal portions of pRB (Fig. 1). We observed that deletion of the majority of the C-terminal half of pRB eliminated transcriptional repression in vivo (Fig. 3). Deletion mutants Gal4-Δ234–921 and Gal4-Δ593–921 exhibited β-galactosidase activity nearly equivalent to or slightly higher than that observed with the DNA binding domain control. Thus, transcriptional repression was shown to reside in the C-terminal portion of the retinoblastoma protein, which contains the pocket domain. We then tested several smaller deletions within the latter half of the molecule in a construct that also eliminated the N-terminal 180 amino acids. In each case, deletion of either the A domain or the B domain individually did not appear to disrupt the ability to repress transcription from the reporter construct. We then tested fusions containing only the A domain or only the B domain and observed that either domain was sufficient to confer transcriptional repression (Fig. 3). Interestingly, however, the A domain alone in the context of the full N terminus was unable to repress transcription (Fig. 3). This may have been due to the lower level of expression of this fusion (Fig. 4) or it may be that the N-terminal 180 amino acids influence the conformation or modification of the A domain in this context. All three deletion mutants tested (Gal4-Δ180,303–421, Gal4-Δ180,622–714, Gal4-Δ180,773–909) exhibited strong transcriptional repression from the reporter construct to ∼20% of the control level (Fig. 3). Indeed, these mutants were stronger repressors than the wild type fusion construct perhaps because in addition to being deletion mutants they might also be defective for phosphorylation.

We verified by Western blot that the various constructs were indeed expressed in yeast. We observed expression of Gal4-p130 and Gal4-pRB (Fig. 4); however, we were unable to demonstrate expression of Gal4-p107 (Fig. 4) and Gal4-Δp34 (data not shown). The fact that we observed transcriptional repression with these latter constructs suggests that they are indeed expressed but that they are either rapidly degraded or inefficiently extracted in our extract preparations. We also observed expression of all of the pRB deletion mutant fusions, although Gal4-Δ593–921 was expressed at relatively lower levels than the other mutants (Fig. 4).

We also tested the transcriptional properties of the known pRB phosphorylation site mutant, Δp34 (40). As shown in Fig. 3 the Δp34 fusion was a stronger repressor than the wild type pRB fusion inhibiting transcription to 20% of control levels. This effect is similar to that observed for the same construct in a mammalian system (19). To address the issue of cyclin dependence of transcriptional repression in vivo, we examined the repression properties of Gal4-pRB, Gal4-Δp34, and Gal4-

FIG. 1. Schematic representation of Gal4 DNA binding domain (DBD, aa 1–147) and various DBD fusion constructs to the pRB family of proteins. Black dots represent cyclin dependent kinase consensus phosphorylation sites that have been mutated in the Δp34 construct.
p130 in yeast strains mutant for individual G1 cyclins. Fig. 5A shows that in strains deleted for either CLN1 or CLN2 there is no effect on transcriptional repression by Gal4-pRb, Gal4-p107, or Gal4-p130. However, in the CLN1CLN2cln3 background we observed a derepression to nearly control levels with all three fusions. These results show that CLN3 is required for transcriptional repression in vivo.

In mammalian cells transcriptional repression by pRb appears to involve a direct interaction with histone deacetylase (HDAC1), which physically interacts with the pocket domain of pRb (23–25). Therefore, we analyzed the requirement for histone deacetylase in repression by Gal4-pRb and Gal4-p130 using a yeast strain mutant for the yeast HDAC1 homolog RPD3. As shown in Fig. 5B, repression by both Gal4-pRb and Gal4-p130 was abolished in the RPD3 deletion strain thus showing a dependence on histone deacetylase function in vivo. Furthermore, repression by Gal4-pRb and Gal4-p130 was also observed to be dependent on the RPD3 cofactor, SIN3 (Fig. 5B).

**DISCUSSION**

The retinoblastoma protein is a transcriptional regulatory molecule whose ability to repress transcription of E2F dependent genes is intimately connected with its ability to regulate the cell cycle. Repression of transcription by pRb is thought to be mediated in two ways. Firstly, the binding of pRb to the activation domain of E2F1 precludes the interaction of this domain with other factors such as TBP or TFIIH, which are required for activation of transcription (44). Secondly, the retinoblastoma protein may directly repress transcription, and the interaction with E2F1 serves solely to recruit pRb and associated factors to appropriate genes (21). We tested the transcriptional capacity of the Rb protein family in yeast using fusions of pRb, p107, and p130 proteins to the heterologous DNA binding domain of yeast Gal4 and showed that all three pRb-family members repress transcription of a given reporter gene simply by virtue of its being tethered to an appropriate DNA binding site in the promoter region.

Repression by Gal4-pRb, Gal4-p107, and Gal4-p130 was absolutely dependent on the presence of a Gal4 binding site in the reporter construct. Furthermore, we tested the repression effect of Gal4-pRb, Gal4-p107, and Gal4-p130 using two lacZ reporter constructs that differed in the position of Gal4 binding sites relative to Gcn4 binding sites. We observed a similar level of repression for all fusion proteins irrespective of whether the

**FIG. 2.** A, β-galactosidase assay of the transcriptional repression function of Gal4 DNA binding domain fusions to the retinoblastoma family of proteins, pRb, p107 and p130, using the lacZ reporter construct pGG9. B, β-galactosidase assay using the alternative lacZ reporter construct, pSS38–3. C, β-galactosidase assay using a reporter construct, pSS36, without Gal4 binding sites. All data are the result of at least three independent transformants assayed in triplicate.

**FIG. 3.** β-galactosidase assay using various pRb deletion constructs fused to Gal4 DBD. All data are the result of at least three independent transformants assayed in triplicate.

**FIG. 4.** Western blot analysis of yeast extracts containing Gal4 DBD alone or Gal4 fusion constructs. 100 μg of total yeast extract are loaded per lane (left panel), except Gal4-Δ593–921 (300 μg), Gal4-Δ180,603–909 (400 μg), and Gal4-Δ592 (500 μg).
binding sites were placed upstream or downstream of the Gcn4 binding sites. This data indicates that the repression effect is unlikely to be a matter of steric hindrance but rather an effect on activated transcription. It is possible that the inhibition of activated transcription by Gal4-pRb is perhaps the result of pRb binding to the activation domain of Gcn4. Such an interaction would presumably result in the inhibition of activated transcription by preventing the binding of factors such as the Swi/Snf complex to the activation domain of Gcn4 (45). Alternatively, the retinoblastoma protein may itself sequester the Swi/Snf complex (26–28) and thereby inhibit Gcn4-dependent transcription. Previous studies have shown that in mammalian cells LexA-pRb can inhibit transcription by activators whose binding sites are located adjacent to the Lex binding site in a reporter construct (20).

In an attempt to define the domains within the retinoblastoma protein important for transcriptional repression we assayed several deletion constructs for their ability to repress transcription from the reporter construct. We showed that the N-terminal region cannot repress transcription from the reporter when fused to the Gal4 DNA binding domain. Analysis of deletions throughout the C-terminal half of the molecule showed that either an intact A or B domain is sufficient for transcriptional repression in vivo. This result is subtly different from that seen in mammalian cells in which any disruption of the pocket domain eliminates transcriptional repression. In experiments that systematically examined the contribution of A and B domains to the repression function of pRb in mammalian cells, it was observed that any disruption of the B domain completely eliminated repression whereas deletion of the A domain retained some residual ability to repress transcription (22). Our results suggest that in yeast, the A and B domains can function independently of each other to repress transcription. Since the A and B domains have been shown to bear some similarity to TBP and TFIIIB (3, 4), they may inhibit transcription by competing for factors/sites that interact with these general initiation factors. The difference in domain requirements for repression between yeast and mammalian data may be related to the fact that in yeast we have used a chromosomally integrated reporter whereas in mammalian cells transient transfection using plasmid-borne reporter genes were assayed (19–22). Additionally, in pRb phosphopeptide analysis, yeast cells do not show phosphorylation of two spots normally seen to be hyperphosphorylated in mammalian cells (18). Since phosphorylation has been shown to affect intramolecular interactions within the retinoblastoma protein (46), it is possible that some aspects of pRb function will be different in yeast as a result of this difference. A recent study shows that an intact LXCXE binding domain may not be essential to some aspects of pRb function in yeast (47).

To address specific aspects of biological function related to the mechanism of pRb repression in yeast we examined the role of phosphorylation, G1 cyclins, and histone deacetylase components in repression by pRb family members. In mammalian cells, the hypophosphorylated pRb mutant, Gal4-Δp34, is a stronger transcriptional repressor than the corresponding wild type fusion (19). In yeast the Gal4-Δp34 fusion protein was also a stronger transcriptional repressor in vivo than the wild type Gal4-pRb fusion protein (Fig. 3). Our studies show that a CLN1CLN2cln3 mutant strain showed reversal of transcriptional repression by Gal4-pRb, Gal4-Δp34 and Gal4-p130 in yeast. No effect on repression was observed in the CLN1cln2cln3 or the cln1cln2cln3 strain backgrounds. The results from the three strain backgrounds suggest that Cln3-dependent phosphorylation is required for repression in vivo. The fact that repression by Gal4-Δp34 was also dependent on CLN3 suggests that Cln3-dependent phosphorylation of sites outside of those mutated in the Δp34 construct are required for repression in vivo. The hypophosphorylated form of pRb has been shown in both yeast and mammalian cells to have almost the same pattern of phosphopeptides present as in the hyperphosphorylated form (18, 48). Therefore, it is not unreasonable that Cln3-dependent phosphorylation could be required for repression. Alternatively, Cln3-dependent phosphorylation of a target factor could be required for repression in vivo. In human cells, coexpression of cyclin E, cyclin B, or cyclin D with Gal4-pRb caused a reversal of transcriptional repression in vivo (19, 46). Analogous experiments using cyclin knockouts in mammalian cells have not been done, and therefore it is difficult to directly compare our data with results obtained with cyclin overexpression experiments.

Since at least one mechanism of repression by pRb is via recruitment of HDAC1 (23–25) we tested the requirement for the yeast HDAC1 homolog RPD3 in repression by Gal4-pRb and Gal4-p130. Repression by both constructs was abolished in the RPD3 deletion strain (Fig. 5B). This data corroborate the requirement for HDAC1 observed in mammalian cells, as well as a recent report showing that Gal4-pRb requires RPD3 for repression in yeast (47). In addition, we observed that repression by Gal4-pRb and Gal4-p130 in yeast was also dependent...
on the RPD3 cofactor, SIN3. The data of Kennedy et al. (47) differ from our studies in the requirement for SIN3 in yeast, and this may be due in part to differences in strain background or the type of assay used to assess function i.e. growth on synthetic drop-out media-His versus β-galactosidase assays.

Our results show that the retinoblastoma family of proteins functions as direct transcriptional repressors in vivo in S. cerevisiae. The domain requirements are subtly different from that observed in mammalian cells in that either the A or B subdomain appears sufficient to mediate transcriptional repression. However, as is the case in mammalian cells, hypophosphorylated versions of pRb exhibit stronger transcriptional repression relative to the wild type protein. We have also shown an effect of pRb, p34, and p130 in yeast, as well as a requirement for the histone deacetylase components, RPD3 and SIN3. Future studies should allow us to make use of both biochemical and genetic approaches to further define the mechanism of direct transcriptional repression by the retinoblastoma protein in vivo.

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Transcriptional Repression by pRb in Yeast
The Retinoblastoma Family of Proteins Directly Represses Transcription in \textit{Saccharomyces cerevisiae}

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