

ABSTRACT

Firstly, the analysis of *nrg1* shows that Nrg1, which is involved in the regulation of glucose repression of the SUC2 and GAL genes of *S. cerevisiae*; specifically, three downstream targets of signaling for glucose: Nrg1, Mig 1, and Mim2.

INTRODUCTION

The role of S-adenosylmethionine (SAME) in glucose metabolism is one of the most important and important topics in bioenergetics. In the context of metabolic syndrome, a number of studies have shown that SAME is required for glucose repression of S-adenosylmethionine (SAME) in *Saccharomyces cerevisiae* (SCC), however, the role of SAME in glucose metabolism has not yet been elucidated.

In this study, we have investigated the role of S-adenosylmethionine (SAME) in the regulation of SCC glucose metabolism in SCC-expressing mice. By using transgenic mice expressing SCC-specific SUC2 and G Yeast *Saccharomyces cerevisiae* uses glucose as its primary carbon source, but when glucose is present in growth media, transcription of several genes responsible for the metabolism of alternative sources of carbon, including GAL, SUC2, MAL and STA genes, is suppressed. Mig1, a zinc-finger protein that binds to DNA consensus sites in GC-rich cores and flanking AT sequences, plays inverse role in glucose repression at several genes. It is hypophosphorylated in high glucose and localized in the nucleus, while phosphorylation in low glucose regulates its activity and transports it to the cytoplasm for phosphate removal. Despite the fact that MIG1 only partially relieves glucose repression at promoters such as SUC2, deletion of either SSN6 or TUP1 completely abolishes it, and the STA1 gene of *S. cerevisiae* var. diastaticus, which is also repressed by glucose, is not affected by mig1. Consequently, other proteins in this category, such as Mig2 (which has sequence similarity to Mig1), play a minor role in glucose suppression in their zinc finger regions of their genes despite selectively. In *S. cerevisiae* var. diastaticus, NRG1, an uncharacterized gene, was required for glucose repression of the STA1 gene through anelid homeostasis (LexA-Nrg1), which is shown to function similarly to a type A reporter construct dependent on both glucose and Ssn6 or Tup1. There has been extensive research on the SUC2 gene's involvement in glucose repression in *S. cerevisiae*. SUC2 has two Mig1 binding sites in its regulatory region and high glucose levels are regulated by nucleosomes that bind to it, while low glucose results in both mig1. However, the exact relationship between these two pathways is not clear. Many genes known as SNF (Sucrose Non-Fermenting) have been identified through genetic screens and are essential for the regulation of SUC2 transcription in the absence of glucose. Genetic analyses and subsequent studies have divided these genes into two groups: one that encodes Snf1 and its associated regulator Snd4, which are required to antagonize the repressive effects of Mig1. The other group is composed of members of the Swi/Snf complex that remodel nucleosomes in an ATP dependent manner. In this work, we report that Nrg1 is now recognized as a key genetic screen for new regulators of SUC2 transcription, and shows that at these genes, Mig1, Mig2, and Nrg1 are redundant for mediating glucose repression by glucose.

CONCLUSION

Remarkable conclusions According to the results, it has been determined that Nrg1 is likely responsible for recruiting the Ssn6/Tup1 complex. The

study concludes that this suggests NrG1 functions as a third repressor, which is necessary for glucose repression at SUC2 and the GAL genes.