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ORIGINAL PAPER

# Biotechnological properties of distillery and laboratory yeasts in response to industrial stresses

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**Abstract** The stress sensitivity of different wild-type strains was evaluated, as well as the response of cells arrested at different cell cycle positions to high hydrostatic pressure (HPP). HHP was chosen both for its importance in food decontamination and assessment of its suitability as a model for stress in general and understanding the yeast stress response. Studies were conducted with four industrial strains and four laboratory wild-type yeast strains (two haploid and two diploid) that differed in their backgrounds. Fundamental differences were found between the laboratory and industrial populations. Industrial strains were clearly more sensitive to hydrostatic pressure and ethanol stresses than the laboratory strains. However, ethanol production was higher in industrial strains than laboratory strains. Furthermore, no correlation was observed between ploidy and stress resistance. Yeast cells arrested in the G1 phase led to an enhancement in pressure tolerance compared to unarrested, G2 arrested, and S arrested cells. Moreover, cells arrested in the S phase were more sensitive to hydrostatic pressure than cells arrested in the G2 phase. Again, industrial strains were more sensitive than laboratory strains. HHP responses of industrial yeasts correlated well with both ethanol concentration and temperature stress, which suggests that it would be a useful model stress.

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**Keywords** Cell cycle · Hydrostatic pressure · Laboratory and industrial strains

**Abbreviations**

| | |
|---|---|
| HHP | High hydrostatic pressure |
| EC | Ethanol stress |
| HS | High temperature treatment |
| OD | Optical density |
| YEPD | Yeast extract, peptone and glucose |
| YPA | Yeast extract, peptone and sodium acetate |
| SF | Sinefungin |
| HU | Hydroxyurea |
| BN | Benomyl |
| HSP | Heat shock protein |

## Introduction

Baking and brewing industries rely upon the ability of *Saccharomyces cerevisiae* to convert glucose into ethanol and carbon dioxide. *S. cerevisiae* has been chosen over the centuries for being physiologically adapted to the multiple stress conditions imposed during the fermentation process. Also, *S. cerevisiae* has been widely used as a model system to study the physiological effects of stresses in microorganisms [25].

The main physical stresses yeasts face are temperature, ethanol concentration, pressure, desiccation, acidity, or alkalinity, osmotic and ionic stress, and low oxygen levels [5]. An abrupt change in the environmental conditions leads to a rapid adjustment of metabolism, transcriptional profile, and development to adapt to the new situation [29, 50, 55].

By far the most studied stresses in living cells are changes in temperature [2, 14]. Nevertheless, research on the effects of exposure of microorganisms to high hydrostatic pressure (HHP) has grown in the last decade, highlighting two issues [40]. The first one is the biotechnological application of HHP. It was reported that pressures up to 10 MPa increase both the rate and the final yield of ethanol production by *S. cerevisiae* cells, and pressure has also been used as a tool to improve yeast biomass yield [12, 45]. In addition, HHP has been applied instead of heat treatment as a decontaminant in food processing industries.

The second issue is the understanding of how organisms adapt to HHP. Pressure is a physical parameter that can cause stress to organisms, with effects related to those of other stresses, such as temperature, ethanol, and oxidative stresses. Thus, it is a particularly useful tool for the understanding of a variety of physicochemical processes that are commonly imposed upon yeast cells in the fermentation process, e.g., osmotic temperature and ethanol stresses. Besides, HHP can be considered a controlled, accurate, and reproducible stress system, causing change only in one variable, the reaction volume, so that the results are easier to interpret than those obtained by varying the temperature, which involves both volume and thermal changes [39].

Information on the nature of pressure stress responses for *S. cerevisiae* has increased in the last two decades [1, 26]. Nevertheless, some discrepancies exist in published data. The nature of the survival curve and the sensitivity of different strains to high hydrostatic pressure still have to be fully characterized. Furthermore, experiments usually are performed with cells in the exponential phase, and even though cells are in the same metabolic stage, they are in different cell cycle phases. Thus, it is important to understand the influence of the cell cycle position on pressure resistance.

Proliferation of all cells is mediated through the cell division cycle that consists of four main phases: genome duplication (S phase) and nuclear division (mitosis or M phase), separated by two gap phases (G1 and G2). At the end of G1 phase, cells decide whether to commit to cell division in a process called START in yeast, while in G2 phase, the transcriptional factors promote entry into mitosis [8, 30]. Cell cycle progression is associated with marked fluctuations in the transcriptional activity of many yeast genes [52]. Stress in yeast cells leads to G1 arrests due to lack of protein kinase A activity, hence inducing a protection response [21]. Previous reports showed that cell cycle- or age-synchronized yeast cultures in G1 phase present increased resistance to heat shock [46], UV radiation [34], and hydrogen peroxide [27] stress compared to asynchronous cultures. However, it has also been reported that the cell cycle phase has no direct consequence for yeast stress resistance [9], and that other characteristics, such as growth rate, would be more important for cell susceptibility [20].

The aim of this work was to extend the knowledge about *S. cerevisiae* survival when submitted to different stress conditions. In this study, comparison between wild-type (wt) laboratory and industrial yeast strains that differed in their genotypic backgrounds was performed in order to monitor their survival response to hydrostatic pressure, ethanol, and temperature treatment. Ethanol production by the same set of cells was determined. In addition, the sporulation efficiency of the industrial strains was measured as a function of stress. The response to pressure in unsynchronized and synchronized yeast cultures was also analyzed with the aim of elucidating the role of the cell cycle phase in *S. cerevisiae* piezoresistance.

## Materials and methods

### Strains and growth conditions

*Saccharomyces cerevisiae* distillery strains, identified by biochemical tests as previously described [10] and a molecular method based on the amplification and sequential analysis of the region D1/D2 of the 26S rDNA domain [16], were isolated as described in Pataro et al. [44]. These strains were collected from fermentation vats of two "cachaça" (Brazilian spirit) distilleries in the State of Espirito Santo, Brazil. Laboratory wild-type and industrial strains used in this work are described in Table 1.

**Table 1** *Saccharomyces cerevisiae* yeast strains used in this work

| Laboratory (background) | Industrial (source) |
|---|---|
| Y440 (*MAT* a, *leu2*) | WST0501 (commercial brewing strain—AEB Bioquímica Latino Americana Ltda, Brazil) |
| BY4741 (*MAT* a, *his3-1, leu2, met15, ura3*) | FF122 (commercial baker's strain—AB Brasil Indústria e Comércio de Alimentos Ltda, Brazil) |
| S288C (*MAT* α, *SUC2, gal2, mal, mel, flo1, flo8-1, hap1*) | BT0502 (Brazilian distillery) |
| W303-1A (*MAT* a, *leu2-3, 112 trp1-1, can1-100, ura3-1, ade2-1, his3-11,15*) | BT0504 (Brazilian distillery) |

Genes deletions are in lower-case italic letters

Cells were grown at 28°C with aeration in liquid YEPD medium (1% yeast extract, 2% peptone, 2% glucose) to exponential phase ($OD_{600\ nm} = 1.0$), and a new culture stock was used for each batch. Yeast cell viability was determined by plating the appropriate dilution of cells on YEPD plates. After incubation for 2 days at 28°C, cell survival was determined by comparing the colony forming units (CFU) of treated versus untreated samples.

## Stress conditions

Yeast cells were submitted to the following stress treatments:

1. HHP was achieved using laboratory equipment adapted from high pressure cells developed by Itskevich [33]. Samples were placed inside a 4-ml Teflon tube and pressurized (50–200 MPa) for 30 min in the absence of air bubbles at room temperature (25 ± 2°C). External hydrostatic pressure was applied to the BeCu piston cylinder and a calibrated mechanical manometer was used to measure it.
2. Ethanol stress was performed by incubating yeast cultures on YEPD supplemented with 15% ethanol for 120 min at 28°C, with aeration (agitation at 150 rpm). Erlenmeyer flasks were tightly closed to avoid ethanol evaporation.
3. High temperature treatment was performed at 45°C for 120 min with aeration.

## Fermentative test and ethanol production

For fermentative capacity analysis, wild-type laboratory and industrial yeast strains were pre-grown in YEPD at 28°C with aeration to stationary phase. Approximately $1.0 \times 10^8$ cell/ml was inoculated in sterile sugarcane juice at 28°C. Time point samples were taken at 0, 4, 24, 48, and 72 h, and centrifuged at $1{,}000{\times}g$ for 2 min. Potassium dichromate method was used to determine ethanol production as described by Vicente et al. [54].

## Sporulation efficiency

Industrial yeast strains were grown overnight in YEPD; diluted 1:200 in YPA (1% yeast extract, 2% peptone, and 2% sodium acetate) and grown to the exponential phase ($OD_{600nm} = 1.0$). Cells were then washed in distilled water and resuspended in 1% sodium acetate (sporulation medium) and incubated for 7 days at 28°C with aeration. Sporulation was analyzed by using an optical microscope, cells and spores were counted by using a Neubauer chamber [31].

## Cell cycle inhibitor treatment

Yeast cells at the exponential phase were incubated at 28°C with agitation (150 rpm) in the presence of a cell proliferation inhibitor. After each treatment, cells were submitted to HHP and analyzed for viability. Non-pressurized samples were used as controls.

Sinefungin (Sigma–Aldrich Brasil Ltda, Brazil) at a final concentration of 1.42 µg/ml and benomyl (DuPont do Brasil S.A., Brazil) at a final concentration of 40 µg/ml were prepared in dimethyl sulfoxide (DMSO, Merck S.A., Brazil). Hydroxyurea (Sigma–Aldrich Brasil Ltda, Brazil) was prepared in distilled water to a final concentration of 6 mg/ml. Figure 1 presents the periodic events of the cell division cycle and arrows point to the specific phase that each drug acts in arresting yeast cells.

The percentage of G1, S, and G2 cells was determined by counting under an optical microscope using a Neubauer counting chamber.

## Statistical analysis

All experiments were performed in duplicate, reproduced at least three times, and mean values were calculated.

Statistical analysis for sporulation efficiency was performed by using commercial software (SPSS version 10) and significance was defined as $P < 0.05$. The univariety analysis was carried out by Pearson correlation to determine whether there was correlation between ploidy efficiency and stress resistance [18].

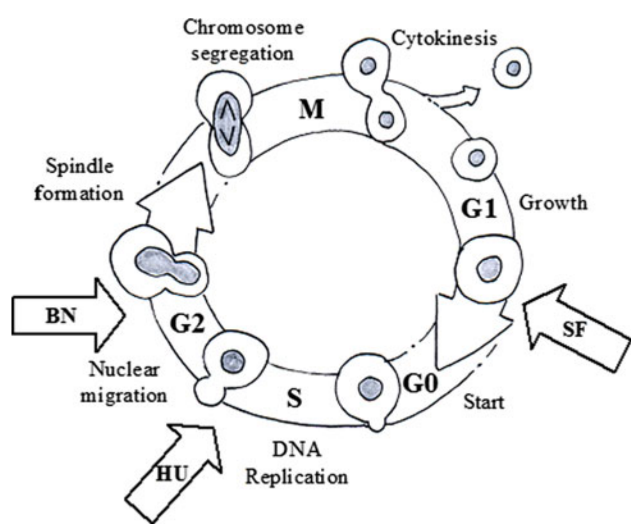


**Fig. 1** Schematic representation of the *S. cerevisiae* cell cycle with *arrows* pointing at G1, S, and G2 phases at which sinefungin (*SF*), hydroxyurea (*HU*), and benomyl (BN) arrest yeast cells, respectively

## Results and discussion

### High hydrostatic pressure response

Wild-type (wt) laboratory and industrial strains were submitted to high hydrostatic pressure (HHP) treatment at room temperature revealing a typical pressure stress survival pattern, as shown in Fig. 2. The model that best fitted the cell survival to HHP followed the random nature of cell killing. The mode of death observed following HHP treatment resembles necrosis [13], as cell membranes are the early targets and organelles are deformed or disrupted [23]. The curve shown in Fig. 2 is based on the single hit and multiple target approach and a simple equation captures the relationship between the applied pressure and cell viability:

$$Y = 100\left(1 - \left(1 - e^{(-P/Po)}\right)^{n}\right) \quad (1)$$

where $Y$ is the fraction of cells that survive a given pressure value, $P$ is the applied pressure, $n$ and $P$o are adjustable constants using a reduced Chi-Sqr fitting of data points.

The results pointed to a universal survival distribution, separated into two groups: wt laboratory strains forming one group and industrial strains another (Fig. 2). Industrial strains (open symbols) were more sensitive to HHP than laboratory strains (filled symbols), clearly observed at pressure values between 50 and 100 MPa. Table 2 was constructed by using the above equation and the data showed that the pressure value necessary to kill 50% of yeast cells was 59–72 and 39–52 MPa for laboratory and industrial strains, respectively, thus confirming that the industrial strains were more piezosensitive than the laboratory strains.

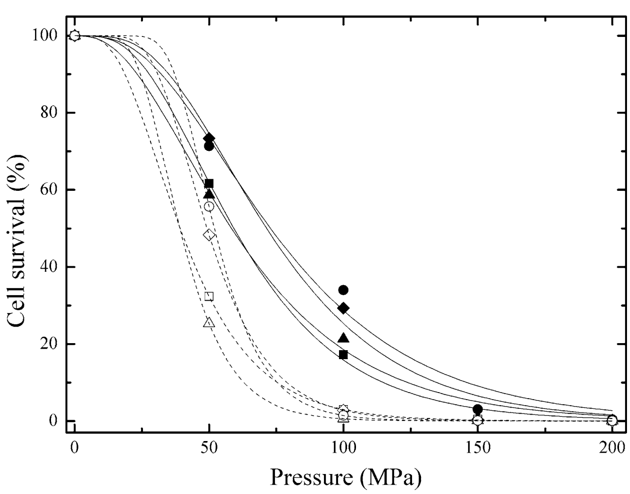


**Fig. 2** *Saccharomyces cerevisiae* cells survival to high hydrostatic pressure stress. Wild-type laboratory (*filled square* Y440, *filled triangle* BY4741, *filled diamond* S288C, *filled circle* W303) and industrial (*open square* WST0501, *open triangle* FF122, *open diamond* BT0502, and *open circle* BT0504) yeast strains in logarithmic phase (1.0 × $10^7$ cells/ml) were submitted to high hydrostatic treatment for 30 min

**Table 2** Pressure value necessary to kill 50% of yeast cells

| | Strains | Pressure values (MPa) |
|---|---|---|
| Laboratory | Y440 | 59.3 |
| | BY4741 | 58.3 |
| | S288C | 71.4 |
| | W303 | 72.8 |
| Industrial | WST0501 | 39.4 |
| | FF122 | 39.2 |
| | BT0502 | 49.3 |
| | BT0504 | 52.0 |

At 50 MPa, at least 60% of each wt laboratory strains survived while the survival rate ranged between 55 and 25% in industrial strains (Fig. 2). The difference between the groups was more evident with increasing pressure. Between 18 and 30% wt laboratory cells were still alive at 100 MPa while only 0.5–3% industrial cells survived at this pressure value. Therefore, although 100 MPa may be considered as a mild stress ( $\sim$50% survival) to laboratory strains, it was a severe stress ( $\sim$0.5% survival) to industrial strains.

The piezosensitivity of wt laboratory strains was evident at pressure values above 100 MPa. W303 (filled circles) had a higher piezotolerance than the other strains and this was clearly seen at 150 and 200 MPa. At 150 MPa around 3% of these cells were still alive whereas all the other strains presented less than 1% of cell survival (Fig. 2).

FF122 (triangle open symbols), a commercial baker's strain, was the most piezosensitive strain and even at 50 MPa only 25% of cells were alive (Fig. 2).

Garay-Arroyo et al. [28] reported that industrial strains showed higher survival rates when compared to laboratory strains under oxidative and heat shock stresses, while for osmotic and freezing stresses, no difference was observed. They proposed that the constant and broad environmental changes to which industrial strains are exposed may be responsible for increasing their stress tolerance. For HHP stress the inverse phenomena was observed and piezotolerance was higher in laboratory strains than industrial strains (Fig. 2). The differences in the strains' genotypic background and in the stress conditions analyzed can partially explain this discrepancy. Miura et al. [41] showed that the loss of one heat shock protein (HSP) gene can enhance cell viability against HHP by up to fivefold. They proposed that the loss of one of the *HSP* genes may induce expression of other *HSP* genes, resulting in piezotolerance. In our results, laboratory strains have deletions on several genes, as shown in the Table 1, and the loss of one gene may induce a different expression profile and/or activation of biochemical pathways responsible for the higher piezotolerance observed in laboratory strains compared to industrial strains. In the same way, a great number of genes not directly related to the stress response were up- or downregulated after pressure treatment on yeast laboratory strain Y440, suggesting an intricate stress response system where other factors may be related to cell survival upon stress conditions [24]. Our results reinforce the importance on characterizing each strain response pattern in order to compare data results and, furthermore, to explore their biotechnological potential.

## High hydrostatic pressure (HHP), ethanol (EC), and heat shock (HS) tolerance of different yeast strains

In order to compare the degree of correlation stress injury effects, a plot correlating the groups of strains (laboratory—filled symbols versus industrial—open symbols) and tolerance to different stress agents (e.g., EC versus HHP) was constructed (Fig. 3). In this plot, the straight line indicates the result if the stresses were equivalent and correlated ($Y = X$). Moreover, these plots allowed the analysis of the stress resistance profile characteristic to each group.

The tested stresses were: temperature, 45°C for 120 min; pressure, 150 MPa for 30 min; and 15% ethanol for 120 min. These were chosen to promote a significant reduction of yeast cell survival. Also, temperature and ethanol are common stresses in the fermentation vats and pressure is used as a model tool for stress resistance.

The proximity of industrial cell results (open symbols) to the correlation line indicates that they respond in a similar manner for each of the different evaluated stresses. The stress response for industrial strains was similar for the three tested treatments, as they were all close or on the line, even though among them different stress sensitivity was observed (Fig. 3a–c). On the other hand, laboratory strains (filled symbols) did not follow the correlation line, showing that these stresses act differently on the cells. Actually, they had a higher resistance to ethanol and to pressure than to heat (Fig. 3a–c). Also, laboratory strains were more tolerant of ethanol and pressure than the industrial strains (Fig. 3a), but they responded similarly to the industrial strains to high temperature, varying from $10^3$ to $10^4$ cells/ml that survived (Fig. 3b–c).

If a greater stress resistance is related to the fermentative environment, one would expect that the wt laboratory strains tested would be more efficient than the industrial ones. Nevertheless, after 72 h of sugarcane juice fermentation, ethanol production by laboratory strains was less than 4% while for industrial strains it was around 8% (Table 3); except for the S288C laboratory strain that had an ethanol production performance similar to the industrial strains, around 7.5% after 72 h of fermentative process. S288C has a deletion on the HAP1 gene, the product of which (Hap1 or Cyp1 protein) is a positive regulator of the cytochrome *c* genes [15]; thus, in the absence of this gene, the respiratory pathway can be impaired and the fermentative pathway is activated.

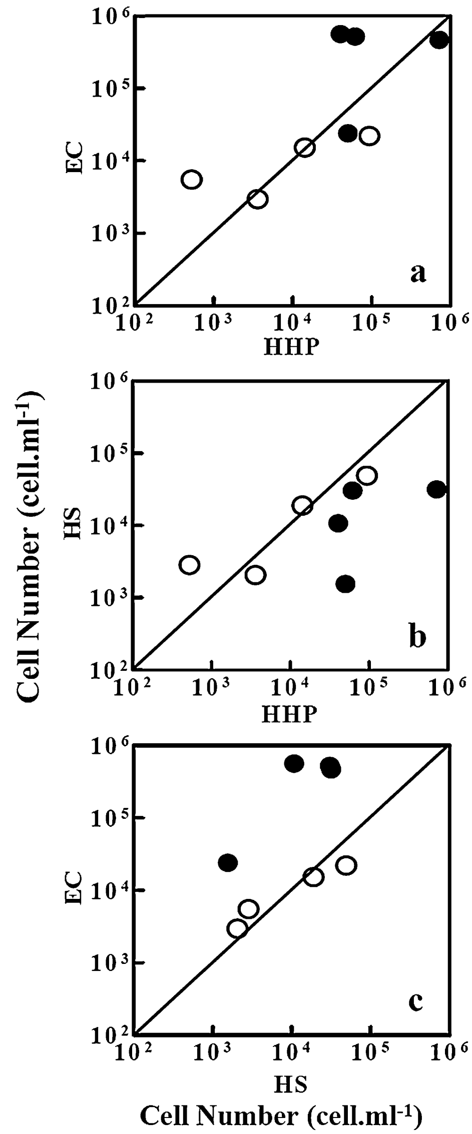


**Fig. 3** Comparative cell survival to high hydrostatic pressure, ethanol, and heat shock stress among laboratory and industrial strains. **a** Correlation of tolerance to ethanol × hydrostatic pressure, **b** heat shock × hydrostatic pressure, and **c** ethanol × heat shock among laboratory (*filled circles*) and industrial (*open circles*) strains. *Numbers* on both axes represent cell survival (cell/ml) after 150 MPa for 30 min (HHP), 15% ethanol for 120 min (EC), or 45°C for 120 min (HS)

Aneuploidy and polyploidy may confer advantages in adapting to a variable external environment due to the gain of extra sets, or incomplete sets, of chromosomes [11, 48]. Nevertheless, no difference has been detected between the

**Table 3** Ethanol production by *Saccharomyces cerevisiae* yeast strains in 72 h of fermentative process

| | Strains | Ethanol (%) | | | |
|---|---|---|---|---|---|
| | | 4 h | 24 h | 48 h | 72 h |
| Laboratory | Y440 | 0 | 1.6 | 3.5 | 3.9 |
| | BY4741 | 0 | 0.6 | 2.2 | 2.9 |
| | S288C | 0 | 2.2 | 6.2 | 7.6 |
| | W303 | 0 | 0.2 | 1.5 | 3.9 |
| Industrial | WST0501 | 0.7 | 4.4 | 7.6 | 8.2 |
| | FF122 | 1.2 | 4.3 | 7.9 | 8.2 |
| | BT0502 | 0.4 | 4.3 | 7.6 | 7.4 |
| | BT0504 | 0.5 | 6.0 | 7.5 | 8.1 |

haploid or diploid laboratory strains when submitted to elevated concentrations of glucose, NaCl, ethanol, lactate, and low pH [3].

Cell ploidy was determined by sporulation efficiency analysis [31]. Sporulation counts from two haploid (BT0504 and FF122) and two polyploid (WST0501 and BT0502) industrial strains (data not shown) were entered into correlation analysis against the stresses tested. There was a weak positive correlation between sporulation efficiency and all tested stresses. These findings suggested an absence of correlation between ploidy and temperature ($r = +0.410$), high pressure ($r = +0.198$), and ethanol ($r = +0.637$, $P = 0.363$) resistance. We suggest that other characteristics beyond genomic duplication are important to stress response analyzed in this work.

## Cell cycle phase and HHP response

Considering that yeast cells may exhibit a distinct tolerance to stress according to their cell cycle position [46] and yeast cells in stationary phase are more resistant to high hydrostatic pressure than proliferating cells [22], we studied the yeast response to HHP treatment at different phases of the cell cycle. For this purpose, the eight different strains were incubated with different cell cycle inhibitors and submitted to HHP treatment.

Figure 4 shows the survival curve of laboratory and industrial strains submitted to 150 MPa for 30 min in the presence or absence of cell cycle inhibitors.

Yeast cultures were treated with sinefungin (SF) for 12 h, at which point over 90% of yeast cells were G1 arrested, and therefore unbudded. SF is a structural analog of *S*-adenosylmethionine [36] that blocks the yeast cell cycle initiation step START. Cells that were exposed to G1-arresting inhibitor (SF) showed a higher tolerance to pressure stress than untreated cells. Also, G1 arrested (SF treated) cells showed a piezotolerance enhancement when compared to S and G2 phase arrested cells (Fig. 4). This

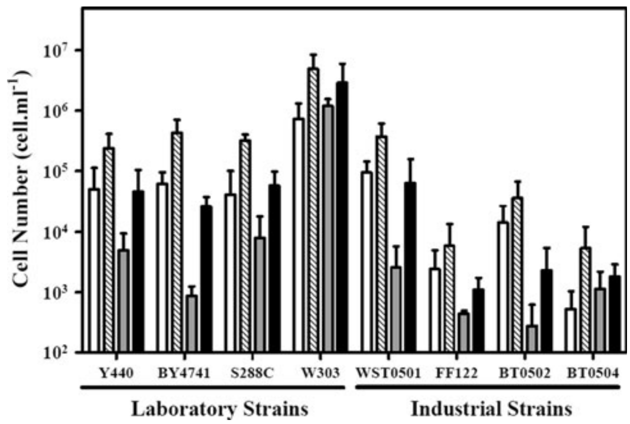


**Fig. 4** Effect of hydrostatic pressure on yeast strains at different cell cycle phases. **a** *S. cerevisiae* cells growing in rich media to the early exponential phase ($1.0 \times 10^7$ cells/ml) were pre-treated with sinefungin, i.e., G1 arrested (*striped bars*), hydroxyurea, i.e., S arrested (*squared bars*), and benomyl, i.e., G2 arrested (*black bars*), and pressurized at 150 MPa for 30 min. As a control, cells incubated without cell cycle inhibitors (*white bars*) were used

result is in agreement with the observations of Rosin and Zimmerman [47], who demonstrated that the survival of a diploid strain (NCYC239) after pressure treatment (100 MPa, 4 h) was enhanced among G1 and G2/M phase cells compared to S phase cells. This is also true to high temperature stress, as unbudded cells are more resistant to heat shock than other cell cycle fractions (budding cells) [46].

Cells treated with hydroxyurea (HU) are S phase arrested, as HU inhibits DNA replication and division in *S. cerevisiae* [51]. In this phase, cells have begun cell division, showing small buds. Yeast strains were treated with HU for 5 h, affording 85% of cells with small buds. S phase arrested cells were more piezosensitive than G1 and G2 phase cells, except that W303 and BT0504 were unresponsive to this treatment (Fig. 4). Sensitivity heat shock has already been observed under HU treatment. S phase arrested cells have previously been shown to be sensitive to the lethal effects of 5-min incubation at 52°C [9]. The appearance of small buds in *S. cerevisiae* cells is related to thermosensitivity [49] and piezosensitivity [47]. The change in DNA conformation during cell cycle [49] and inhibition of the DNA repair system [47] are pointed to as the major factors responsible for the higher sensitivity of S phase yeast cells to HS and HHP. Since DNA molecules are particularly resistant to HHP [38], we believe that other factors may be responsible for the enhanced piezosensitivity of S phase cells. Most likely, changes in yeast ultrastructure during the cell cycle may be sufficient to modify the piezotolerance of yeast cells. Indeed, the oriented growth in *S. cerevisiae* cells is mediated by a polarized actin cytoskeleton that directs secretory vesicles to the growth or bud site [17]. When cells are pressurized, the actin cables in mother cells disappear and the cell cycle-specific actin organization is lost at 100 MPa. Short and thick fragmented actin cables are seen in both buds and mother cells at 150 MPa [35]; thus, cells in the bud phase can be more sensitive than other arrested cells.

Benomyl (BN) causes disruption of the microtubule cytoskeleton during the cell cycle [32] and was used as a G2 phase inhibitor. In this phase, cells are characterized by bud growth and achromatic fuse formation [56]. BN was added to yeast cells for 4 h, leading to 90% of G2 arrested cells. BN treated cells did not show any piezosensitivity difference when compared to untreated cells (Fig. 4). DNA microarray analyses of yeast cells responding to benomyl showed expression of *YAP1*, a gene that encodes for a transcriptional factor known for its role in the regulation of the response to oxidative stress [37]. Similarly, it was demonstrated that HHP induces genes related to oxidative stress, such as *CTT1* and *SOD1* [24], and that pretreatment with hydrogen peroxide induces piezotolerance in yeast [43]. HHP in *Schizosaccharomyces pombe* can affect actin monomers and tubulin dimers [4]. As mentioned above, HHP treatment on *S. cerevisiae* cells induces alterations on actin organization at various cell cycle phases [35]. Also, disruption of the microtubular network causes dissociation of the Golgi cisternae both in *S. pombe* and *S. cerevisiae* [7, 23]. The fact that benomyl treated and untreated cells display a similar response can be justified because benomyl binds to tubulin structure sites, which participate directly in cytokinesis [19]; therefore, the synergistic effects of HHP and benomyl on the tubulin structure of *S. cerevisiae* may constitute a stress that compensates for the results of oxidative stress.

Individual cells within clonal microbial cultures exhibit marked phenotypic heterogeneity. Such heterogeneity is evident in the differential sensitivity to stress of genetically identical cells, and can be fundamental to the fitness and persistence of an organism [6, 52, 53]. When synchronized isogenic cells in the culture are submitted to stress treatment, one could expect that all cells in the culture would respond homogeneously. However, the heterogeneity of cells due to cell ageing, mitochondrial activity, epigenetic regulation, and stochastic variation [42] produce heterogenic resistance, even within genetically identical populations grown under uniform conditions. This phenotypic heterogeneity may in part explain the differences observed among the yeast strains tested under HHP.

In summary, in this work a universal survival model was suggested for different *Saccharomyces cerevisiae* yeast strains when submitted to high hydrostatic pressure treatment. Even though all strains had a decrease in cell survival while pressure values increased, the survival pattern was clearly separated into two groups of yeast strains, laboratory and industrial. Moreover, cell cycle position also had a significant effect on yeast sensitivity to pressure. This paper provides a valuable insight into the properties of industrial yeasts vital to the brewing, baking, and ethanol biotechnology industries, in comparison to laboratory strains, and their responses to a model stress, pressure.

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