The *Saccharomyces cerevisiae* YDL219w (*DTD1*) gene, which codes for an amino acid sequence sharing 34% identity with the *Escherichia coli* D-Tyr-tRNA_{Tyr} deacylase, was cloned, and its product was functionally characterized. Overexpression in the yeast of the *DTD1* gene from a multicopy plasmid increased D-Tyr-tRNA_{Tyr} deacylase activity in crude extracts by two orders of magnitude. Upon disruption of the chromosomal gene, deacylase activity was decreased by more than 90%, and the sensitivity to D-tyrosine of the growth of *S. cerevisiae* was exacerbated. The toxicity of D-tyrosine was also enhanced under conditions of nitrogen starvation, which stimulated the uptake of D-amino acids. In relation with these behaviors, the capacity of purified *S. cerevisiae* amino acid dehydrogenases in bacteria (2, 3) or of D-amino acid oxidases in various eucaryotes (4–7). In the case of fungi, a homolog of the *E. coli* dtd gene in several other bacteria as well as in the yeast *Saccharomyces cerevisiae*, in the nematode *Caenorhabditis elegans*, in the higher plant *Arabidopsis thaliana*, in mice, and in man. Such a ubiquitous character suggests the hydrolysis of D-Tyr-tRNA_{Tyr} to be a universal mechanism of defense against a harmful effect of D-tyrosine. However, before drawing such a conclusion, more cells have to be examined for the occurrence of a relationship between D-Tyr-tRNA_{Tyr} hydrolysis and protection against D-tyrosine.

In the present study, we functionally characterize the *S. cerevisiae* YDL219w gene, which codes for a protein showing 34% identity with the *E. coli* D-Tyr-tRNA_{Tyr} deacylase. The YDL219w gene was amplified by PCR and cloned. Expression of this gene from a multicopy plasmid increased D-Tyr-tRNA_{Tyr} deacylase activity in crude extracts, whereas disruption of the chromosomal YDL219w gene decreased deacylase activity and exacerbated the sensitivity of *S. cerevisiae* to D-tyrosine. We propose the name of *DTD1* for the YDL219w gene.

**MATERIALS AND METHODS**

Brewe’s yeast tRNA was from Roche Molecular Biochemicals. L-[14C]tyrosine acceptance of this tRNA was 23 pmol/μg. Q-Sepharose was from Amersham Pharmacia Biotech. Nickel-nitrilotriacetic acid-agarose was from Qiagen. D-Methylene[3H]tyrosine (211 GBq/mmol) was custom prepared by Amersham Pharmacia Biotech. L-[14C]tyrosine (18.4 GBq/mmol) was from NEN Life Science Products. Gentamicin sulfate and unlabeled L- and D-tyrosine were from Sigma. Plasmid pYES2/GS-YGR185CY was from Invitrogen. YPD medium was made as described before (21). Yeast nitrogen base without amino acids and ammonium sulfate, from Difco, was used for the preparation of minimal media.

**Cloning of the DTD1 Gene—**Genomic DNA of *S. cerevisiae* strain DBY2057 (Table I) (22) was prepared by the procedure of Hoffman and Winston (23). Then, the *DTD1* gene of *S. cerevisiae* was amplified by PCR using 0.1 μg of this DNA plus oligonucleotides CGGGGATCCGATTTACAAATGAGATTTGCTTACAAAAAAGTC and GCTCTAGAGTCATCTTTAGTCGTCAGTTCGAAGAATGG as primers. The PCR fragment of expected size (549 base pairs) was purified using the Qiagen PCR purification kit-50, digested by *Bam*HI and *Xho*I, and inserted into the corresponding sites of plasmid pYES2 to give the plasmid pYES2-DTD1.

The nucleotide sequence of the cloned *DTD1* gene differed from the sequence in the genomic data base by a C→T substitution located 293 bases downstream from the A of the ATG initiation codon. This change did not modify the amino acid sequence of the protein produced. The plasmid pYES2-DTD1 was used to transform the yeast strain DBY2057 by the lithium acetate method (24).

**Disruption of the DTD1 Gene—**Disruption of the *DTD1* gene was performed by the PCR-based method of Wach *et al.* (25) using the *kanMX* cassette as a selectable marker. This cassette contains the its product accounts for more than 90% of the deacylase activity in crude extract. However, although the *dtd* gene is essential to afford protection against the toxicity of D-tyrosine added to the culture medium, it does not interfere with cell growth under standard conditions (20).
kanamycin resistance of the E. coli transposon Tn903 fused to transcriptional and translational control sequences of the *Ashbya gossypii* *TEF* gene. Consequently, *S. cerevisiae* transformants are efficiently selected because of their acquired resistance to Geneticin (G418) (26). A DNA fragment containing the *kanMX* cassette from plasmid pFA6-kanMX4, flanked by 40 base pairs of the target locus, was amplified by PCR using oligonucleotides: AGCCAAGCATCTGTAGTCGT-CTTCATTAGTTAAAGAGCAACTCATCATTGCGCCGAATATCGATG-AATTCGAGCTCG.

Plasmid pYES2-DTD1 from Invitrogen contains the *His6* epitope tag and a polyhistidine tag (His6). The nucleotide sequence of pYES2-DTD1 was digested by *Sal* I, *Stu* I, and recircularized. Using the above procedure, the His<sub>6</sub>-tagged tyrosyl-tRNA synthetase was purified approximately 140-fold when compared with the crude extract. It was homogeneous according to SDS-PAGE analysis. Concentration of the His<sub>6</sub>-tagged tyrosyl-tRNA synthetase was determined using a *M*<sub>2</sub> of 2 × 47,712 and a light absorption coefficient of 0.439 A<sub>47,712</sub> units mg<sup>-1</sup> ml<sup>-1</sup> as deduced from the amino acid sequence.

**Enzymatic Assays**—Measurements of initial rates of δ-Tyr-tRNA<sup>δTyr</sup> deacylase activity were performed in crude extracts as described earlier, using *E. coli* δ-[<sup>14</sup>Cl]tyrosine as substrate (20). One unit of enzyme activity corresponds to 1 pmol of δ-Tyr-tRNA<sup>δTyr</sup> hydrolyzed/min.

δ-Tyr-tRNA synthetase activity was assayed during 10 min at 28 °C in 100 μl of a reaction mixture containing 20 mM Tris-HCl (pH 7.8), 75 μCi of [3H]tyrosine, 10 mM 2-mercaptoethanol, 1 mM phenylmethylsulfonyl fluoride, 0.5% bovine serum albumin, 2.5 mM 2-mercaptoethanol, 5.3 mg/ml crude brewer's yeast tRNA, catalytic amounts of tyrosyl-tRNA synthetase, and indicated amounts of δ-[<sup>14</sup>C]tyrosine (500 Ci/mmol) or δ-[<sup>3H</sup>]tyrosine (500 Ci/mmol). The reaction was quenched by the addition of (i) 2.5 ml of ice-cold trichloroacetic acid (5%, w/w) containing 0.5% tyrosine and (ii) 10 μl of carrier RNA from yeast (4 mg/ml). The precipitate was recovered on Whatman GP-C filters, and the retained radioactivity was measured in a Beckman LS1801 scintillation counter. One unit of enzyme initial activity corresponds to 1 pmol of tyrosine transferred onto the tRNA/min.

**RESULTS**

**Cloning of the DTD1 Gene**—Comparative analysis using the BLAST program revealed the presence in *S. cerevisiae* of a gene homologous to the *E. coli* *dtd* gene encoding δ-Tyr-tRNA<sup>δTyr</sup> deacylase. This gene, *DTD1* (or YDL219w), is located on chromosome IV and contains one intron of 71 base pairs. To investigate its function and physiological importance, the *DTD1* gene was amplified by PCR and cloned into the yeast expression vector pYES2. In the resulting plasmid (pYES2-DTD1), the *DTD1* gene was placed under the control of the Gal1 portion of the divergent GALI/GAL10 promoter.

Cells DBY2057 transformed by plasmid pYES2-DTD1 were cultivated in minimal medium with galactose as the carbon source, i.e., under conditions inducing full expression of the cloned gene. δ-Tyr-tRNA<sup>δTyr</sup> deacylase activity in a crude extract of the transformed strain was increased 190-fold as compared with strain DBY2057 (Table II). When cells were grown in the presence of glucose, the presence of the plasmid pYES2-DTD1 increased the deacylase activity by a factor of 1.7 only. These results showed that the δ-Tyr-tRNA<sup>δTyr</sup> deacylase activity recovered in crude extracts followed the expected level of expression of the cloned *DTD1* gene.

**Inactivation of the *S. cerevisiae* DTD1 Gene**—To determine whether the DTD1 protein was responsible for the previously reported δ-Tyr-tRNA<sup>δTyr</sup> deacylase activity in yeast crude extracts (17), the *DTD1* gene was disrupted. For this purpose, the chromosomal gene of the *S. cerevisiae* haploid strain DBY2057 was interrupted by the *kanMX* cassette using a PCR-based procedure.

The *DTD1* gene inactivation led to a more than 10-fold decrease in the δ-Tyr-tRNA<sup>δTyr</sup> deacylase activity in the crude extract (Table II). Remarkably, however, the inactivation did not affect the growth rate of yeast cells in rich YPD medium or minimal yeast nitrogen base medium.

**δ-Tyrosine Toxicity**—The δ-steroisomers of various amino acids (δ-histidine, δ-methionine, δ-serine, δ-phenylalanine, δ-
leucine, β-alanine, β-tryptophan, and β-tyrosine) inhibit the growth of wild-type *S. cerevisiae* cells (28). These β-amino acids are imported into yeast cells by the general amino acid permease corresponding to the *GAP1* gene (28, 29). The activity of this permease is controlled through a double mechanism that involves derepression of the *GAP1* gene expression under limiting nitrogen conditions, on the one hand, and inactivation of the permease activity by ammonium ions, on the other hand. As a result, the toxicity of β-amino acids is enhanced when cells are grown in a medium devoid of ammonium salts (30–32). Such observations led us to compare the toxicity of β-tyrosine in a minimal medium containing either L-proline or ammonium sulfate as the nitrogen source. Nitrogen starvation is known to induce the filamentous (pseudohyphal) growth of diploids of *S. cerevisiae* (33, 34). Consequently, to avoid filamentation, we used haploid strains.

On minimal medium agar plates supplemented with L-proline, the growths of the wild-type strain (DBY2057) and of the *dtd1* mutant strain (DBY2057ΔDTD1) were identical. Upon addition of 30 μM β-tyrosine, a significant inhibition of the growth of the mutant colonies only was observed (Fig. 1A). At 300 μM β-tyrosine, the colony formation of either strain was strongly inhibited.

On minimal medium agar plates containing ammonium sulfate, the growth of all strains became less sensitive to the presence of β-tyrosine. This behavior reflects the inhibition of the GAP1 permease by ammonium ions (29). However, a relatively stronger effect of β-tyrosine on the growth of the mutant strain (DBY2057ΔDTD1) was visible again (Fig. 1B). Therefore, whatever the culture condition assayed, the *dtd1* strain was more sensitive to β-tyrosine than the parental *DTD1* strain.

To investigate further the relation between the toxicity of β-tyrosine and the expression of the *DTD1* gene, strains DBY2057 and DBY2057ΔDTD1 were transformed by either plasmid pYES2-DTD1 or control plasmid pYES2. An overexpression of the *DTD1* gene harbored by the pYES2-DTD1 plasmid was ensured by the presence of galactose. Under this condition, the growths of strains DBY2057(pYES2-DTD1) and DBY2057ΔDTD1(pYES2-DTD1) became indistinguishable whatever the concentration of β-tyrosine in the solid medium (Fig. 2). In agreement with this observation, levels of overproduced β-Tyr-tRNAβTyrc activity in extracts from the two strains were very similar (Table II). Therefore, the addition of a plasmid-borne functional *DTD1* gene was enough to cure the specific sensitivity of strain DBY2057ΔDTD1 to β-tyrosine and to give this strain the phenotype of DBY2057(pYES2-DTD1). Moreover, because they each overexpress the *DTD1* gene, the two pYES2-DTD1 carrying strains grew slightly better than DBY2057(pYES2) in the presence of β-tyrosine.

On minimal medium agar plates supplemented with glucose, the sensitivity to β-tyrosine of strain DBY2057ΔDTD1 still responded to the transformation with plasmid pYES2-DTD1. However, as clearly shown on the plates containing 30 or 100 μM β-tyrosine (Fig. 3), the strain DBY2057ΔDTD1 transformed by pYES2-DTD1 remained slightly more sensitive to β-tyrosine than the control *DTD1* strain transformed by either pYES2-DTD1 or pYES2 plasmid. This behavior reflects the specific repression of the cloned *DTD1* gene on the plasmid under glucose conditions. Indeed, the intracellular level of β-Tyr-tRNAβTyr deacylase activity derived from plasmid pYES2-DTD1 in the context of strain DBY2057ΔDTD1 was 2-fold smaller than that arising from the chromosome in the wild-type strain DBY2057 carrying control plasmid pYES2 (Table II).

### Table II

| Strain              | β-Tyr-tRNAβTyr deacylase activitya | Glucoseb | Galactoseb |
|---------------------|-----------------------------------|----------|------------|
| DBY2057             | 2,600                             | 2,800    |            |
| DBY2057ΔDTD1        | 150                               | 110      |            |
| DBY2057(pYES2-DTD1) | 4,100                             | 53,000   |            |
| DBY2057(pYES2)      | 2,700                             | 2,800    |            |
| DBY2057ΔDTD1(pYES2-DTD1) | 1,300               | 43,000   |            |
| DBY2057ΔDTD1(pYES2) | 120                               | 140      |            |

* a One unit corresponds to the enzyme activity capable of hydrolyzing 1 pmol of β-[3H]Tyr-tRNATyr/min under standard assay conditions.

* b Yeast cells were grown in minimal medium (yeast nitrogen base without amino acids and ammonium sulfate) supplemented with L-proline (0.5 mg/ml) as nitrogen source and 2% glucose or galactose as carbon and energy source. With nontransformed strains DBY2057 and DBY2057ΔDTD1, uracil was also added to the medium (50 μg/ml). Specific activity of the deacylase was measured in crude extracts obtained by sonication of steady-state cultures (optical density of 7–10 at 650 nm) resuspended at an optical density of 100 at 650 nm.

FIG. 1. Growth of the *S. cerevisiae* wild-type strain (DBY2057) and of the *dtd1* mutant (DBY2057ΔDTD1) in the presence of various concentrations of β-tyrosine. Cells were grown on minimal medium agar plates supplemented with uracil, glucose, and either L-proline (A) or ammonium sulfate (B). The concentrations of β-tyrosine added to the medium are indicated (0, 30, and 300 μM). Plates containing ammonium sulfate or L-proline were incubated at 30°C for 2 or 3 days, respectively.

Amiocaetylation of tRNA with β-Tyrosine in *S. cerevisiae*—Altogether, the above results suggested that the *DTD1* gene product can protect *S. cerevisiae* against the toxicity of externally added β-tyrosine through an intracellular hydrolysis of β-Tyr-tRNAβTyr. Such a conclusion implies that misacylation of...
on a Q-Sepharose column. Using crude brewer’s yeast tRNA and radioactive D-tyrosine (3.5 μM) as substrates, the obtained tyrosyl-tRNA synthetase sample could be shown to produce D-tyrosylated tRNA at an initial rate of 4 units/mg of total protein in the assay. With 3.5 μM L-tyrosine, under the same experimental conditions, the rate of tRNA\textsuperscript{TYR}\textsuperscript{3H} aminoacylation was 680 units/mg.

In the second set of experiments, homogeneous His\textsubscript{6}-tagged tyrosyl-tRNA synthetase of \textit{S. cerevisiae} was used for aminoacylation assays. With 1 μM D-tyrosine, the initial rate of tRNA aminoacylation was equal to 0.9 × 10\textsuperscript{-3} s\textsuperscript{-1}. Under the same reaction conditions, the enzyme aminoacylated tRNA with 1 μM L-tyrosine at the rate of 0.13 s\textsuperscript{-1}. Therefore, the ratio of initial rates with L-tyrosine or D-tyrosine measured with tagged protein was comparable to that determined with partially purified native tyrosyl-tRNA synthetase (ratio values of 145 and 170, respectively). Finally, the initial rate of D-Tyr-tRNA\textsuperscript{TYR} formation by tagged tyrosyl-tRNA synthetase in the presence of 1 μM D-[\textsuperscript{3H}]tyrosine was reduced by at least 98% upon addition of 5 μM nonradioactive L-tyrosine to the incubation mixture.

Synthesis of D-Tyr-tRNA\textsuperscript{TYR} by tagged tyrosyl-tRNA synthetase could be further established by aminoacylation assays conducted in the presence of pure \textit{E. coli} i-Tyr-tRNA\textsuperscript{TYR} deacylase (20). Tyrosyl-tRNA synthetase concentration in the assay (270 nm) was adjusted so that within 10 min, 150 nm of D-tyrosylated tRNA was produced in the absence of deacylase. When 150 nm deacylase was present, the production of D-Tyr-tRNA was reduced by more than 96%. In parallel experiments, we verified that the presence of D-Tyr-tRNA deacylase did not affect the formation of L-Tyr-tRNA\textsuperscript{TYR}. All these results reinforce our initial views that mischarging of D-tyrosine onto tRNA\textsuperscript{TYR} by \textit{S. cerevisiae} tyrosyl-tRNA synthetase is at the origin of at least a part of the toxicity of this D-amino acid.

**FIG. 3.** Effect of D-tyrosine on the growth (3 days, 30 °C) of the \textit{S. cerevisiae} strains DBY2057 (wild-type) and DBY2057\textit{DTD1} carrying plasmid pYES2\textit{DTD1} or control plasmid pYES2. To induce full expression of the cloned \textit{DTD1} gene, cells were cultivated on minimal medium agar plates with galactose as a carbon source. L-Proline was used as nitrogen source to favor \textit{GAP1} gene expression. D-Tyrosine concentrations in the growth medium are indicated (0, 10, 30, and 100 μM).

**FIG. 2.** Effect of D-tyrosine on the growth (9 days, 30 °C) of the \textit{S. cerevisiae} strains DBY2057 (wild-type) and DBY2057\textit{DTD1} carrying plasmid pYES2\textit{DTD1} or control plasmid pYES2. To induce full expression of the cloned \textit{DTD1} gene, cells were cultivated on minimal medium agar plates with galactose as a carbon source. L-Proline was used as nitrogen source to favor \textit{GAP1} gene expression. D-Tyrosine concentrations in the growth medium are indicated (0, 10, 30, and 100 μM).

The present study shows that, like \textit{E. coli}, \textit{S. cerevisiae} harbors a gene encoding a protein with D-Tyr-tRNA\textsuperscript{TYR} deacylase activity. This gene confers protection to the cell against one harmful effect of D-tyrosine. Consequently, the phylogenetic distribution of the \textit{dtd/DTD1} homologs may be considered again.

When the available complete genome sequences are examined, it is striking to note that, systematically, the organisms that are auxotrophic for L-tyrosine (\textit{Mycoplasma pneumoniae}, \textit{Mycoplasma genitalium}, \textit{Rickettsia prowazekii}, \textit{Borrelia burgdorferi}, \textit{Treponema pallidum}, \textit{Chlamydia trachomatis}, and \textit{Chlamydia pneumoniae}) lack a \textit{dtd/DTD1}-like gene. Consequently, it is tempting to conclude that those cells, which do not synthesize L-tyrosine, do not produce D-tyrosine and therefore do not need a deacylase activity. This idea implies that D-tyrosine can be made as a side product of the anabolic pathways for L-tyrosine synthesis.

Whatever the considered organism, the biosynthesis of L-tyrosine from prephenate always involves two steps: a decarboxylation-dehydrogenation step and a transamination step (35–37). However, the time sequence of these two steps depends on the cell. In organisms like \textit{E. coli} or \textit{B. subtilis}, the decarboxylation-dehydrogenation of prephenate takes place first. It produces 4-hydroxyphenylpyruvate. The last step of L-tyrosine biosynthesis is the addition of an amino group to the future C\textsubscript{α} atom of L-tyrosine (37). One may assume that, upon this transformation, D-tyrosine can appear as a side product and requires D-Tyr-tRNA\textsuperscript{TYR} deacylase to circumvent the ensuing toxicity.

In other organisms such as most cyanobacteria, the transamination of prephenate is made first leading to L-arogenate. In a second step, L-arogenate is transformed into L-tyrosine by aro-
genate dehydrogenase (35, 38). In this case, the production of D-tyrosine would definitely support the idea of an active metabolic pool of this amino acid in the yeast cell.

From the genome data only, it is difficult, however, to predict which pathway is used for the biosynthesis of L-tyrosine. The reason is that the enzymes of the two pathways can share the production of L-tyrosine via the 4-hydroxyphenylpyruvate (44) among organisms like Serratia, Erwinia, Aeromonas, and Pseudo-

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d-Tyrosyl-tRNA\textsubscript{Tyr} Metabolism in \textit{Saccharomyces cerevisiae}

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\textit{J. Biol. Chem.} 2000, 275:11626-11630.
doi: 10.1074/jbc.275.16.11626

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