

# Comparative Gene Expression Profiles Following UV Exposure in Wild-Type and SOS-Deficient *Escherichia coli*

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Manuscript received October 4, 2000  
Accepted for publication January 29, 2001

## ABSTRACT

The SOS response in UV-irradiated *Escherichia coli* includes the upregulation of several dozen genes that are negatively regulated by the LexA repressor. Using DNA microarrays containing amplified DNA fragments from 95.5% of all open reading frames identified on the *E. coli* chromosome, we have examined the changes in gene expression following UV exposure in both wild-type cells and *lexA1* mutants, which are unable to induce genes under LexA control. We report here the time courses of expression of the genes surrounding the 26 documented *lexA*-regulated regions on the *E. coli* chromosome. We observed 17 additional sites that responded in a *lexA*-dependent manner and a large number of genes that were upregulated in a *lexA*-independent manner although upregulation in this manner was generally not more than twofold. In addition, several transcripts were either downregulated or degraded following UV irradiation. These newly identified UV-responsive genes are discussed with respect to their possible roles in cellular recovery following exposure to UV irradiation.

**I**RRADIATION of growing *Escherichia coli* cultures with ultraviolet light (UV) produces DNA lesions that at least transiently block the essential processes of replication and transcription. A large amount of work has demonstrated that the cell responds to this stress by upregulating the expression of several genes that function to repair the DNA lesions, restore replication, and prevent premature cell division. A number of other genes are known to be upregulated, yet remain functionally uncharacterized. The changes in gene expression in response to DNA damage produced by UV and some other environmental agents have been collectively termed the SOS response, after the international distress signal (RADMAN 1974 and reviewed in FRIEDBERG *et al.* 1995; KOCH and WOODGATE 1998).

Many of the DNA damage-induced genes are negatively regulated by the LexA repressor protein, which binds to a 20-bp consensus sequence in the operator region of the genes, suppressing their expression (BRENT and PTASHNE 1981; LITTLE *et al.* 1981). Derepression of these genes occurs when the RecA protein binds to single-stranded regions of DNA created at replication forks when they are blocked by DNA damage. RecA bound to single-strand DNA becomes conformationally active, serving as a coprotease to cleave the LexA repressor. As the cellular concentration of LexA diminishes,

the genes normally suppressed by LexA are more frequently transcribed (SASSANFAR and ROBERTS 1990 and references therein; FRIEDBERG *et al.* 1995). An interesting feature of the LexA/RecA regulatory circuit is that the timing, duration, and level of induction can vary for each LexA-regulated gene, depending upon the location and binding affinity of the LexA box(es) relative to the strength of the promoter. As a result of these properties, some genes may be partially induced in response to even endogenous levels of DNA damage, while other genes appear to be induced only when high or persistent DNA damage is present in the cell. In fact, the SOS response may represent a continuum in the monitoring of environmental stress, rather than simply operating as an emergency switch following acute injury.

The first systematic search for *damage-inducible (din)* genes was carried out by KENYON and WALKER (1980) by randomly inserting a *lac* reporter gene into the *E. coli* chromosome to identify promoters that were upregulated following DNA damage in a *recA/lexA*-dependent fashion. Using this same technique, subsequent studies identified additional *din* genes and in some cases identified genes previously characterized to be involved in the recovery from DNA damage (BAGG *et al.* 1981; FOGLIANO and SCHENDEL 1981; HUISMAN and D'ARI 1981; KENYON and WALKER 1981; SHURVINTON and LLOYD 1982; LLOYD *et al.* 1983; SIEGEL 1983; BONNER *et al.* 1990; IWASAKI *et al.* 1990; OHMORI *et al.* 1995b). Analysis of the known *din* genes revealed a 20-bp consensus LexA-binding motif, or "SOS box," shared by these genes in their promoter/operator regions (WALKER 1984), which has been used in more recent studies to systemati-

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cally search and identify additional *lexA*-regulated genes (LEWIS *et al.* 1994; OHMORI *et al.* 1995a; FERNANDEZ DE HENESTROSA *et al.* 2000). These studies in total have identified 31 genes under *lexA/recA* control.

Other genes have been reported to be upregulated following DNA damage but are believed to be independent of the *lexA* regulon. In some cases, the induction is thought to be dependent on *recA*, but independent from the LexA repressor. In other cases, genes have been shown to be upregulated independently from both *recA* and *lexA* (FRIEDBERG *et al.* 1995; KOCH and WOODGATE 1998). The mechanism of regulation in these cases is not understood. An additional, although as yet unexplored, possibility is that some genes may be repressed or their transcripts may be degraded in response to DNA damage.

It was of interest to us not only to learn whether additional genes can be regulated in a LexA-dependent manner but also to determine whether other cellular responses to UV irradiation exist that are *lexA* independent. The *lexA1* allele encodes an amino acid change at a position that is essential for the cleavage and inactivation of LexA (SLILATY and LITTLE 1987). Thus, in *lexA1* mutants, the LexA1 concentration remains high and LexA-regulated genes are not induced, even in the presence of high levels of activated RecA. Therefore this system should allow an analysis of gene expression that occurs independent of the LexA repressor.

The changes in gene expression in the entire genome can be measured simultaneously using high-density cDNA microarrays (SCHENA *et al.* 1995). DNA microarrays contain PCR-amplified DNA fragments of known and predicted genetic sequences that are printed on the surface of a glass slide. Through the comparative hybridization of two cellular RNA preparations, the relative difference between transcript levels of any gene in these preparations can be determined. Using the complete sequence of the *E. coli* genome (BLATTNER *et al.* 1997), DNA microarrays were prepared containing PCR products corresponding to 95.5% (4101 out of 4295) of all annotated open reading frames in the *E. coli* genome (KHODURSKY *et al.* 2000). We utilized these microarrays to follow the changes in gene expression occurring during the first hour following UV irradiation in the wild-type strain, MG1655, and in an isogenic *lexA1* mutant.

## MATERIALS AND METHODS

**Bacteria:** Strain MG1655 was used as the wild-type strain in this study since its genome has been completely sequenced (BLATTNER *et al.* 1997). The MG1655 *lexA1*(Ind<sup>-</sup>) *malB*:Tn9 was constructed by P1-mediated transduction of the *lexA1* allele from strain GC2281 (TADDEI *et al.* 1995) into strain MG1655. Transfer of the *lexA1* allele was verified by resistance to chloramphenicol and hypersensitivity to UV irradiation.

**Growth and irradiation:** Cells were grown in Davis medium plus 0.4% glucose. Cultures were inoculated at a 1:200 dilution

from a fresh overnight culture into 200 ml Davis media and incubated in a 1-liter Erlenmeyer flask at 37° in a New Brunswick Scientific (Edison, NJ) model G76 gyrotory water bath at 220 rpm to midlog (OD<sub>600</sub> 0.4,  $\sim 2 \times 10^8$  cells/ml). A 15-W germicidal lamp (254 nm, 0.66 J/m<sup>2</sup>/sec at the sample position) provided the UV irradiation. A total of 70 ml of culture was placed into a 15-cm-diameter glass petri dish and irradiated for 60 sec with gentle agitation. Two 65-ml unirradiated samples were also agitated in a 15-cm petri dish but were not exposed to UV. A total of 70 ml of irradiated culture (in a 500-ml Erlenmeyer flask) and 30 ml of unirradiated culture (in a 250-ml Erlenmeyer flask) were then returned to the shaking water bath for the duration of the time course. At the appropriate times, 10-ml samples were placed into 20 ml of ice-cold NET (100 mM NaCl, 10 mM Tris, 10 mM EDTA), pelleted by centrifugation, washed with 1 ml cold NET, repelleted, and frozen at -80°. The limited availability of microarray chips constrained this experiment to a single time course containing seven samples (five irradiated, two unirradiated) for each strain.

**Microarray procedures:** Relative mRNA levels were determined by parallel two-color hybridization to cDNA microarrays representing 4101 open reading frames (ORFs) representing 95.5% of *E. coli* ORFs according to BLATTNER *et al.* (1997). cDNA arrays were manufactured as described in MGuide at <http://cmgm.stanford.edu/pbrown/mguide/index.html>. Total mRNA was extracted from  $2-5 \times 10^9$  cells using QIAGEN (Chatsworth, CA) RNeasy spin columns. A total of 25–30 µg of total RNA was labeled with Cy-3-dUTP (or Cy-5-dUTP) in a standard reverse transcriptase (RT) reaction by Superscript II (+) (GIBCO BRL, Gaithersburg, MD) with 1 µg of random hexamer (Pharmacia, Piscataway, NJ) primers. Following purification through Microcon-30 (Millipore, Bedford, MA) (MGuide), Cy-3- and Cy-5-labeled cDNA were combined with SSC (2.5× final), SDS (0.25%), and 40 µg of *E. coli* rRNA (Boehringer Mannheim, Indianapolis) in a final volume of 16 ml and hybridized to a DNA microarray for 5 hr at 65°. Slides were washed as described in MGuide and scanned using an AxonScanner (Axon Instruments, Foster City, CA; GenPix 1.0) at 10 mm per pixel resolution. Acquired 16-bit TIFF images were analyzed using ScanAlyze software, which is publicly available at <http://rana.stanford.edu/software/>.

**Comparative measurements of transcript abundance:** Time course samples were analyzed directly by comparing the abundance of each gene's transcripts relative to the *t*<sub>0</sub> sample. RNA samples taken during the time course were labeled with Cy-5, and RNA from the *t*<sub>0</sub> sample was labeled with Cy-3.

**Sequence analysis:** Nucleotide sequences in the regions of induced genes were examined using the COLIBRI program provided by the Pasteur Institute at <http://genolist.pasteur.fr/colibri/>. Regions surrounding induced genes were searched for the consensus sequence CTG(N)<sub>10</sub>CAG, allowing for one mismatch. Matching sequences that fell between -400 and +100 bp of a start codon were then examined for their heterology index. The heterology index was determined as reported in LEWIS *et al.* (1994) on the basis of the formula developed by BERG and VON HIPPLE (1988). Heterology index =  $\sum \ln[(n_{(\text{consensus})} + 0.5)/(n_{(\text{actual})} + 0.5)]$ , where *n*(consensus) refers to the number of times that the most common, or consensus, base occurs at a given position in the set of known binding sites, and *n*(actual) refers to the number of times that the base being analyzed occurs at the same position in the set of known binding sites. *n* values for each position of the 20-bp LexA binding site were determined using the known LexA-binding sites shown in Figure 1A and their respective complementary sequences.

**Nomenclature:** All genes are named according to the Rudd system at <http://bmb.med.miami.edu/ecogene/ecoweb> (RUDD

2000). In cases where we found no corresponding Rudd gene for the open reading frame examined, the original identification numbers of Blattner, b##### (BLATTNER *et al.* 1997), were used.

**Raw data:** The raw data from these experiments are available for download at the following web address <http://www2.msstate.edu/~jcc129>.

## RESULTS AND DISCUSSION

We examined the response of *E. coli* strain MG1655 following a dose of 40 J/m<sup>2</sup> (254 nm). Previous studies in our laboratory have shown that exposing an exponentially growing culture of *E. coli* to 40 J/m<sup>2</sup> of UV produces approximately one cyclobutane pyrimidine dimer on each strand per 6 kb of DNA (MELLON and HANAWALT 1989; CROWLEY and HANAWALT 1998). This dose transiently inhibits both replication and transcription, and induces a strong SOS response (COURCELLE *et al.* 1997; CROWLEY and HANAWALT 1998). More than half of the cells survive and genomic replication fully recovers within ~45 min following UV irradiation. Within that time, most of the DNA lesions have also been repaired (MELLON and HANAWALT 1989; COURCELLE *et al.* 1999).

To examine the changes in gene expression in response to this dose of UV irradiation, we compared samples of total RNA taken 5, 10, 20, 40, and 60 min after irradiation to samples made just prior to irradiation. To control for UV-independent changes, total RNA preparations from nonirradiated samples at 20 and 60 min were also prepared. This analysis was carried out with the wild-type MG1655 strain as well as the isogenic *lexA1* derivative and represents the changes in transcript levels of each gene from up to seven independent comparative hybridizations for each cell line, which were observed within the same experiment.

The average change in transcript level in the irradiated samples compared to those in the unirradiated samples for each gene along the *E. coli* chromosome is plotted sequentially in Figure 1. In some cases, no data were plotted for a gene, because either the PCR reaction failed during microarray construction or the fluorescent signal in the unirradiated samples was too low for reliable detection. However, raw data for any or all genes are available for downloading at the web addresses indicated in MATERIALS AND METHODS or upon request to the authors.

**Genes induced in a LexA-dependent manner following UV irradiation:** Twenty-six functional LexA-binding regions controlling at least 31 genes have been previously demonstrated to be functionally active following irradiation. At the time at which these bacterial microarrays were constructed, 3 of these genes, *ysdAB*, *dinQ*, and *dinS*, had not yet been identified as open reading frames (FERNANDEZ DE HENESTROSA *et al.* 2000) and were not included in our analysis. The time courses observed for all other genes/operons known to be regulated by LexA and that contain LexA-binding sites are

plotted in Figure 2A. For most of the LexA-regulated genes, the level and timing of the induction observed in our experiments are in good agreement with previous observations. In confirmation of previous studies, we find that *recN*, *recA*, and *sulA* are heavily induced within the first 5 min of irradiation whereas the *uvrD* induction is much less robust (CASAREGOLA *et al.* 1982; ARTHUR and EASTLAKE 1983; SALLES and PAOLETTI 1983; PICKSLEY *et al.* 1984; SANDLER 1994). *umuCumuD* are also known to be strongly induced; however, full induction of these genes is not observed until 20 min after UV irradiation (WOODGATE and ENNIS 1991). We were unable to assay *ftsK* induction in wild-type cells due to a problem amplifying the *ftsK* fragment when constructing the bacterial microarray. However, some *lexA*-dependent induction is observed in *lolA*, possibly representing some transcriptional readthrough from the *ftsK* gene. In some cases, we observed co-upregulation of the neighboring ORFs that are transcribed in the same orientation. This effect can clearly be seen in the induction of *dinB* transcription, which also renders an increase in *yafN*, *yafO*, and *yafP* mRNA. Similarly, *yebF* and *yebE* are also induced along with LexA-regulated *yebG*. In the case of *recN*, the downstream genes *smpA* and *smpB* also appear to be upregulated following UV irradiation. However, for *b2619* and *b2618* it is actually the antisense strand that would be transcribed following UV irradiation if this induction represents transcriptional readthrough. The actual mechanism of such coregulation could be produced by: (1) transcriptional readthrough resulting from inefficient transcriptional termination; (2) the actual operon spanning across the entire group of neighboring genes; or (3) a regional effect conferred through protein factors or DNA structural conformations within the region under consideration.

Some of the transcripts from documented LexA-regulated genes, *dinG*, *molR*, *uvrD*, and *uvrA*, did not significantly rise following UV irradiation. However, in each of these cases, the samples of these transcripts in the unirradiated (control) culture were significantly decreased during the time course. The reason for this observation is unclear. However, both initial and unirradiated samples were “mock” UV treated by gentle agitation for 60 sec in a 15-cm glass petri dish and it is possible that some genes were affected by this treatment. Importantly, however, when comparing the net change in irradiated and unirradiated samples, the *lexA*-dependent induction of these genes is clearly evident: 1.77-, 1.78-, 2.51-, and 3.85-fold increases, respectively.

Of the reported LexA-regulated genes, we did not detect significant induction of either *hokE* or *ssb* in our experiment. *recA/lexA*-dependent transcription from *hokE* has previously been shown to occur in the *E. coli* strain RW118 following mitomycin C treatment (FERNANDEZ DE HENESTROSA *et al.* 2000). However, the SOS induction of *ssb* is less clear. Although a plasmid-encoded *ssb* has been shown to be slightly upregulated



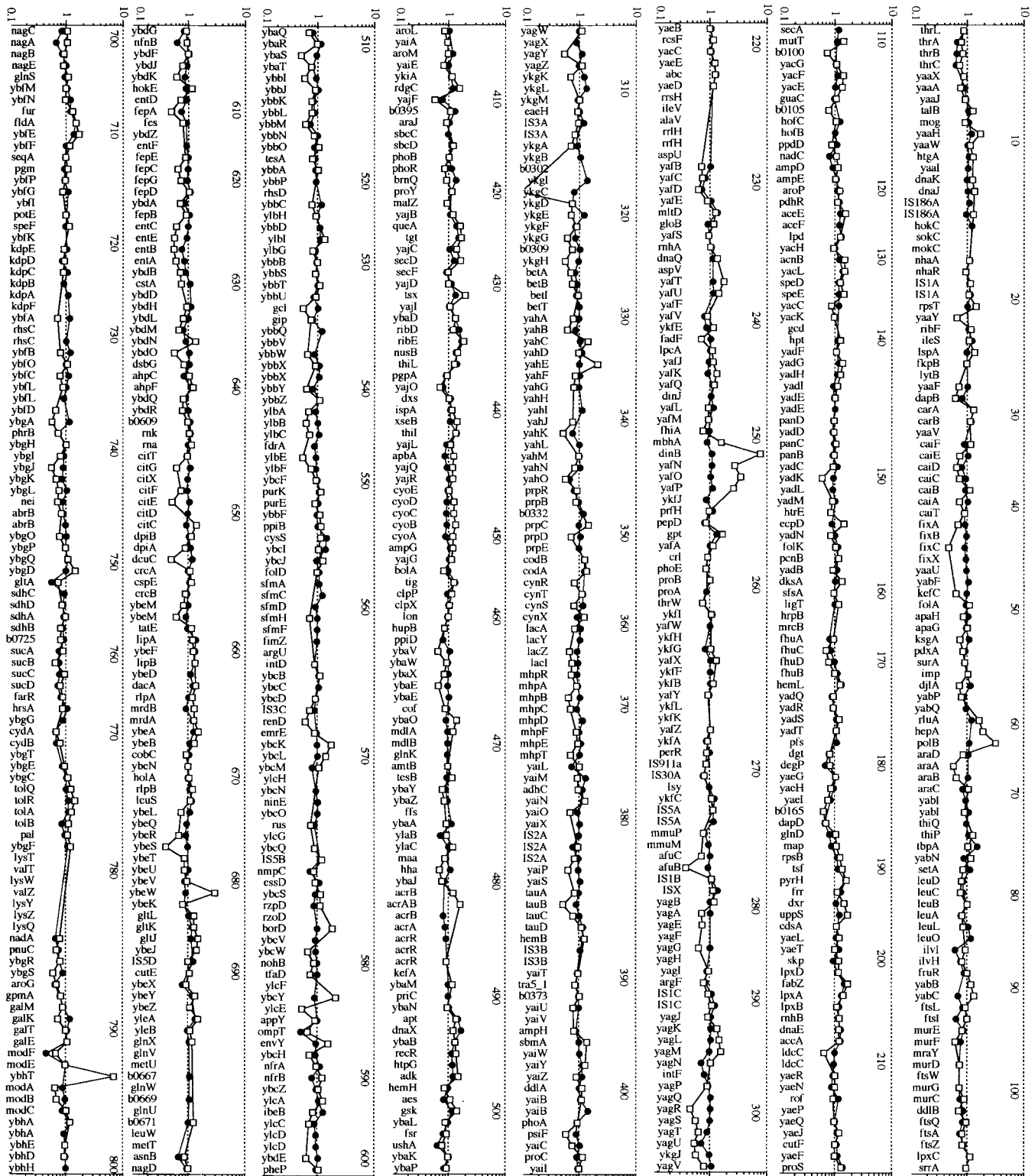


FIGURE 1.—Changes in gene expression within the *E. coli* genome following UV irradiation. The average change in transcript levels in the irradiated samples compared to unirradiated samples for each gene along the *E. coli* chromosome is plotted sequentially. Open squares, wild type; solid circles, *lexA1*. The location of genes on the chromosome, in kilobase pairs, is indicated along the top of each graph. The average change in transcript levels was calculated as the (average change in irradiated samples) / (average change in unirradiated samples). The data for all plotted genes represent an average of between 3 and 5 irradiated time points and at least one unirradiated time point. Time points for which the PCR or hybridization failed, or the fluorescent signal generated by the unirradiated sample was <30% above the background level of fluorescence, were not included in the averages.





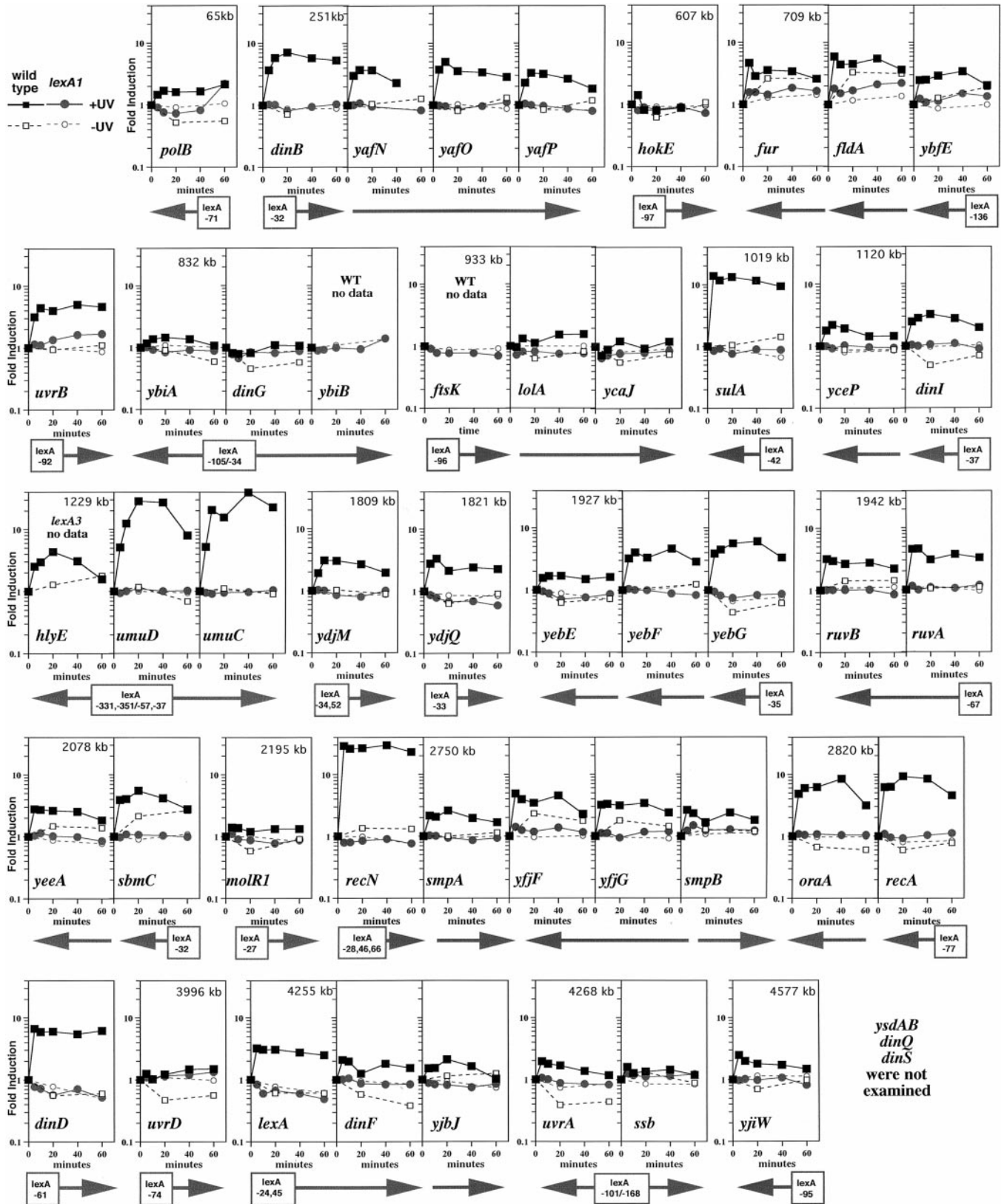












following SOS induction, no induction of the chromosomally encoded *ssb* has been reported and SOS induction does not lead to higher levels of SSB protein (BRAND-SMA *et al.* 1983; PERRINO *et al.* 1987).

If the LexA-binding site is located within the operator regions of two diverging transcripts, it is possible that a single site may regulate the transcription of both operons. This is presumed to be the case at *uvrA* and *ssb* and also appears to occur between *ybiA* and *dinG* as well as *umuCD* and *hlyE*.

In addition to the previously reported *lexA*-regulated genes, we observed several other genes that appeared to be upregulated in a LexA/RecA-dependent manner (Figure 2B). However, to determine whether these genes are directly under LexA control will require further investigation. Some of these genes do appear to have potential candidate LexA boxes (Table 1B). One method of predicting whether a LexA-like sequence will bind LexA is to examine its heterology index (HI), which is a value derived from a mathematical formula ranking the relatedness of a potential sequence to that of known LexA-binding sequences (LEWIS *et al.* 1994). Low HI values predict that a potential sequence is more likely to bind LexA protein. Previous studies found that *lexA* sequences with an HI value of <15 generally bound LexA (LEWIS *et al.* 1994) and recently this method was used to identify seven new *lexA*-regulated genes (FERNANDEZ DE HENESTROSA *et al.* 2000). However, there are exceptions to the predictions from HI values. On the basis of earlier calculations that were based upon a smaller set of *lexA* sequences, FERNANDEZ DE HENESTROSA *et al.* (2000) found that although *dinJ* had an HI value of 7.06, it did not bind LexA. Yet *ybfE*, which they calculated to have an HI value of 14.07, did bind LexA (LEWIS *et al.* 1994). This observation suggests that all factors comprising a functional LexA box have not yet been identified. Therefore, we have recalculated the HI values using all 28 functional LexA sequences found on the chromosome and we have included any potential LexA-binding sequence with an HI value of <20 in Table 1B.

While some of these newly identified genes appear to have potential LexA-binding sequences, many of the induced genes do not, suggesting that in some cases the regulation may occur indirectly. Indirect *lexA*-dependent induction of these genes might occur through regulatory proteins that are themselves under *lexA* regu-

lation, or through other regulatory proteins that are inactivated by a similar RecA-catalyzed proteolysis. The activated form of RecA is also known to induce proteolytic cleavage of other proteins containing "LexA-like" cleavage motifs such as those found in UmuD, the plasmid-encoded MucA, and the repressor proteins of several bacteriophage (LITTLE 1984; PERRY *et al.* 1985). This possibility is especially attractive considering that several of the newly identified genes, *borD*, the *lit-intE* region, and *ogrK*, share homology with cryptic prophage genes. The *borD* gene product is a homolog of the phage  $\lambda$  Bor protein, a lipoprotein expressed during lysogeny that is present in the outer membrane. The *borD* gene product shares homology to other bacterial virulence proteins and its expression increases *E. coli* survival in animal serum (BARONDESS and BECKWITH 1990, 1995).

*lit*, *intE*, and several genes of unknown function in this same region are expressed at relatively late times following UV exposure (Figure 3). *lit* encodes a protease specific for elongation factor EF-tu. Expression of *lit* is induced at late times following phage T4 infection and prevents late phase phage amplification through its EF-tu proteolysis (GEORGIU *et al.* 1998). Following T4 infection, the endogenous *lit* gene has been suggested to trigger an apoptotic-like death of the infected cell, thereby thwarting the reproduction of the virus and precluding the widespread infection of the population. Further characterization is required to know whether this activity or any of the genes in this region affect recovery following DNA damage.

*ogrK* is a prophage gene from a phage P2. *Ogr* has been shown to regulate late P2 gene transcription through an interaction with the host RNA polymerase (WOOD *et al.* 1997). Additionally, an *ogr*-like gene, *regC* in *Serratia marcescens*, has been shown to be induced following mitomycin C treatment in an SOS-dependent manner (JIN *et al.* 1996).

*grxA* is a glutoredoxin that acts as a hydrogen donor for the *E. coli* ribonucleotide reductases. Several thioredoxin and glutoredoxin genes in *E. coli* are coregulated with ribonucleotide reductase gene expression (PRIETO-ALAMO *et al.* 2000). From this perspective, it is interesting to note that the ribonucleotide reductase genes *nrdA* and *nrdB* are among the strongest *lexA*-independent induced genes following UV exposure (Figure 4). *grxA* is also induced in an *oxyR*-dependent manner under some conditions (PRIETO-ALAMO *et al.* 2000).

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FIGURE 2.—Transcriptional induction following UV irradiation in the genes surrounding known LexA boxes. The change in transcript levels for the indicated gene is plotted over time. The arrows indicate the direction of transcription within the operon relative to the LexA box. Arrows pointing left are transcribed on the minus strand and arrows pointing right are transcribed on the plus strand. The locations and distances, in base pairs, of the LexA box from the initial ATG codon are indicated in the boxes. The graphs of genes that are directly adjacent on the chromosome are joined together. The location of the gene(s) on the chromosome, in kilobase pairs, is indicated along the top of each plot. Solid squares, irradiated wild type; open squares, unirradiated wild type; solid circles, irradiated *lexA1*; open circles, unirradiated *lexA1*. Time points in which the PCR or hybridization failed, or the fluorescent signal generated by the unirradiated sample was <30% above the background level of fluorescence, are not plotted.



TABLE 1

## Known and potential LexA boxes surrounding induced genes

A. Known genes	LexA box sequence	HI value <sup>a</sup>
<i>ysdAB</i>	tactgtttatattatcacagta	1.81
<i>umuDC</i>	tactgtatataaaaaacagta	2.12
<i>sbcC</i>	tactgtatataaaaaacagta	2.12
<i>pcsA</i>	aactgtatataaaatcacagtt	2.19
<i>recN</i> no. 1	tactgtatataaaaaacagtt	3.53
<i>dinQ</i>	tactgtatgattatccagtt	3.92
<i>uvrB</i>	aactgtttttttatccagta	4.26
<i>dinI</i>	acctgtataaataaccagta	4.84
<i>hokE</i>	cactgtataaataaacagct	4.92
<i>recA</i>	tactgtatgctcatacagta	4.98
<i>sulA</i>	tactgtacatccatacagta	5.39
<i>uvrA</i>	tactgtatattcattcaggt	6.23
<i>ssb</i>	acctgaatgaatatacagta	6.23
<i>yebG</i>	tactgtataaaatcacagtt	6.26
<i>lexA/denF</i> no. 2	aactgtatatacaccaggg	7.25
<i>ydjQ</i>	cactggatagataaccagca	7.42
<i>lexA/dinF</i> no. 1	tgctgtatataactcacagca	7.45
<i>ruvAB</i>	cgctggatattctatccagca	7.59
<i>yjiW</i>	tactgatgatatacaggt	7.92
<i>molR</i>	aactggataaaaattacaggg	8.14
<i>dinS</i>	agctgtatattgtctccagta	8.24
<i>uvrD</i>	atctgtatatacaccagct	8.46
<i>recN</i> no. 2	tactgtacacaataaacagta	8.50
<i>dinG</i>	tattggctgtttatacagta	8.65
<i>yigN</i>	aactggacgttttgtacagca	8.82
<i>ydjM1</i>	tactgtacgtatcagagtt	9.05
<i>ftsK</i>	tcctgttaatccatacagca	9.18
<i>dinB</i>	cactgtatactttaccagtg	9.40
<i>recN</i> no. 3	taatggttttttcatacagga	10.08
<i>ydjM2</i>	cactgtataaaaaatcctata	10.85
<i>ybfE</i>	aactgattaaaaaccagcg	10.92
<i>polB</i>	gactgtataaaaccacagcc	12.55
Consensus	taCTGTatataatataCAGta	

(continued)

*glvB* induction is unusual in that *glvB* lies in the middle of a predicted operon and encodes a portion of a protein transport system. Nevertheless, *glvB* induction was also observed following exposure to gamma irradiation (data not shown) and may be driven from an alternative promoter or alternative open reading frame in the region.

In the case of *yigN*, a previous study has demonstrated that it contains a functional LexA binding site; however, no further increase in *yigN* expression was observed following treatment with mitomycin C in wild-type, *lexA51* (deficient), or *lexA1* (uninducible) *E. coli* cultures. We have no clear explanation of this difference. However, alternative promoters proximal to *yigN* could have allowed for full expression to occur in these previous studies prior to mitomycin treatment since *yigN* appeared to be heavily expressed under all conditions in that study (FERNANDEZ DE HENESTROSA *et al.* 2000).

TABLE 1

## (Continued)

B.	Potential LexA box	HI value <sup>b</sup>	Bases from start
<i>intE</i> region			
<i>intE</i>	ggctgctgaaaaatacagaa	16.94	-195
<i>ymfI</i>	ttctgtaccagaaaacagtt	15.48	84
<i>ymfM</i>	agctgcaggagcatgcagca	19.32	-122
<i>lit</i>	tgatgacagagtgtccagtg	20.32	-193
<i>ymfE</i>	cactggacactctgtcatca	20.32	-280
<i>intE</i>	ggcgggtataagcatccagtg	14.76	84
<i>intE</i>	tgctgaaaaatacagaagta	20.81	-192
<i>ymfM</i>	ggcagttattcaaacagat	19.98	-222
<i>ymfM</i>	aaccgcatgagaagacagca	18.91	-173
<i>ymfN</i>	aactgattgcgcttccctgta	16.89	-312
<i>ymfN</i>	cgctggttcaaagatcacta	20.90	-152
<i>ymgF</i> region			
<i>ymgF</i>	cggtgtaattatagacagct	15.08	-105
<i>ymgH</i>	aactgaaaaaactccccggg	19.13	6
<i>ydeO</i> region			
<i>ydeO</i>	aaatgcatgcgaccacagtg	20.68	-272
<i>ydeT</i> region			
<i>ydeS</i>	tactgaaccagcagacagca	16.79	-43
<i>yneL</i>	cactgcatacgaaacacca	18.21	-57
<i>yoaA</i> region			
<i>yoaB</i>	ccctggttgatttgaacaggg	13.32	-123
<i>yoaA</i>	ccctggttcaaatcaacaggg	13.32	-24
<i>ogrK</i> region			
<i>ogrK</i>	cattgtcctttatgccagca	19.29	8
<i>ogrK</i>	gactggacaatcactaaggt	19.35	-193
<i>yqgC</i> region			
<i>yqgC</i>	acatggattttccagcagtg	18.78	-193
<i>yqgC</i>	ctcagtaactgtaaccagct	20.65	-41
<i>yhiL</i> region			
<i>yhiL</i>	atctgtttttcagacaagta	18.22	-63
<i>yhiL</i>	tgctgttggtttttacaatt	12.30	-187
<i>glvB</i> region			
<i>glvG</i>	tcctgaagtgccattcagcg	17.45	211
<i>glvG</i>	taatgaccaaattctcagtg	19.17	0
<i>glvB</i>	tgctggtgggaattaccgaa	20.04	-174
<i>glvC</i>	ggctggccaaaagtacaaat	20.85	578
<i>glvC</i>	tgctgtcggtttaccattg	15.97	214
<i>ibpA</i> region			
<i>ibpA</i>	tgctgaaaaataacatcatca	17.25	-249
<i>yigN</i> region			
<i>yigN</i>	aactggacgttttgtacagca	8.82	-61

<sup>a</sup> HI values were calculated from all sequences reported by LEWIS *et al.* (1994) and FERNANDEZ DE HENESTROSA *et al.* (2000). The HI values reported here were calculated to include the results of FERNANDEZ DE HENESTROSA *et al.* (2000) and therefore differ from the values used in their previous study.

<sup>b</sup> No sequences with HI values <20 were found for *grxA*, *borD*, *ybiN*, *arpB*, *yccF*, or *yifL*.

*ibpA* (*hslT*) and *ibpB* (*hslS*), encoding heat-inducible chaperonins, were also induced in a LexA-dependent manner.

There has been little functional characterization of the remaining induced genes. *yoaA* shares homology

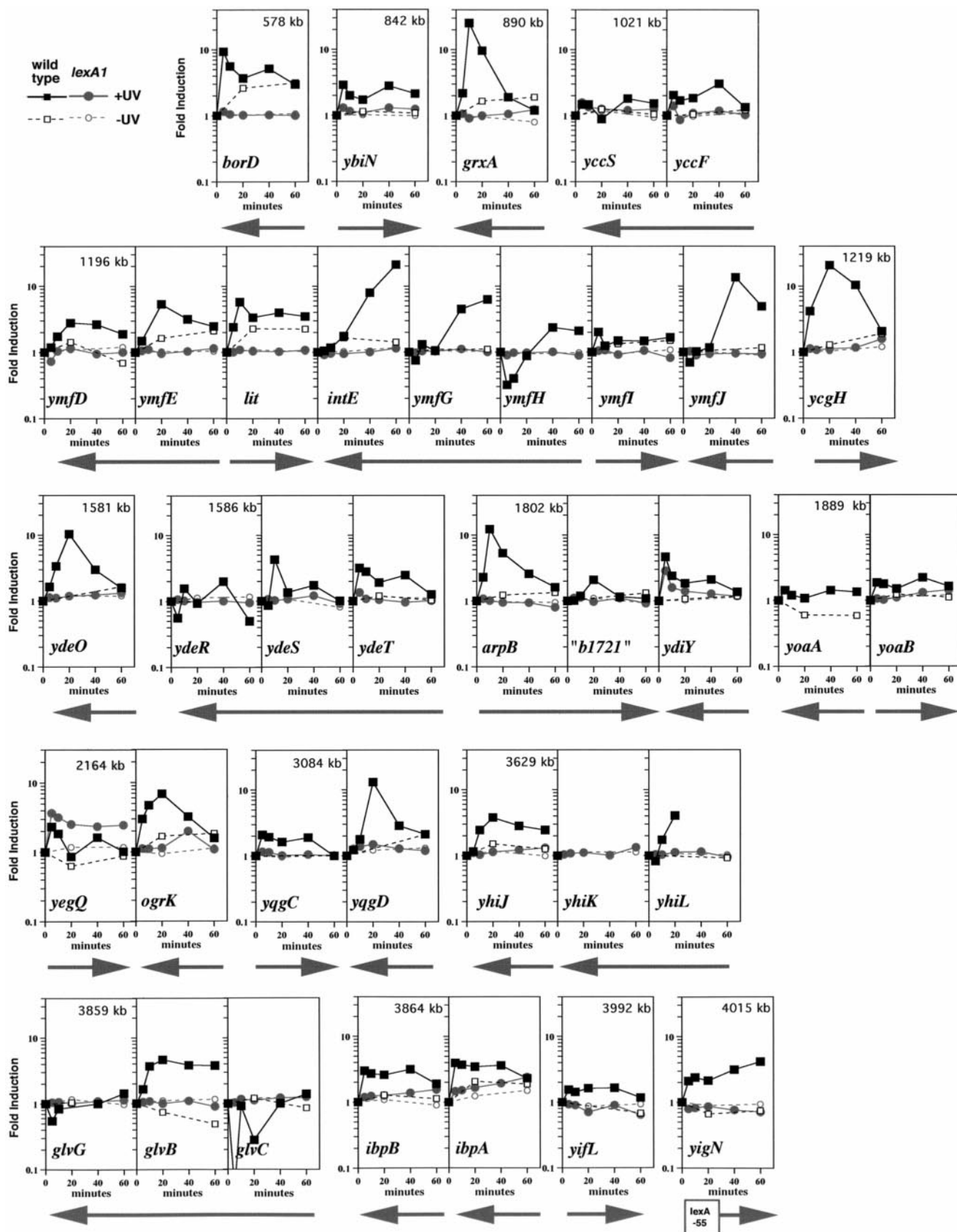


FIGURE 3.—Genes that displayed the largest LexA-dependent transcriptional induction following UV irradiation are plotted. The change in transcript levels for the indicated gene is plotted as in Figure 2. Solid squares, irradiated wild type; open squares, unirradiated wild type; solid circles, irradiated *lexA1*; open circles, unirradiated *lexA1*.

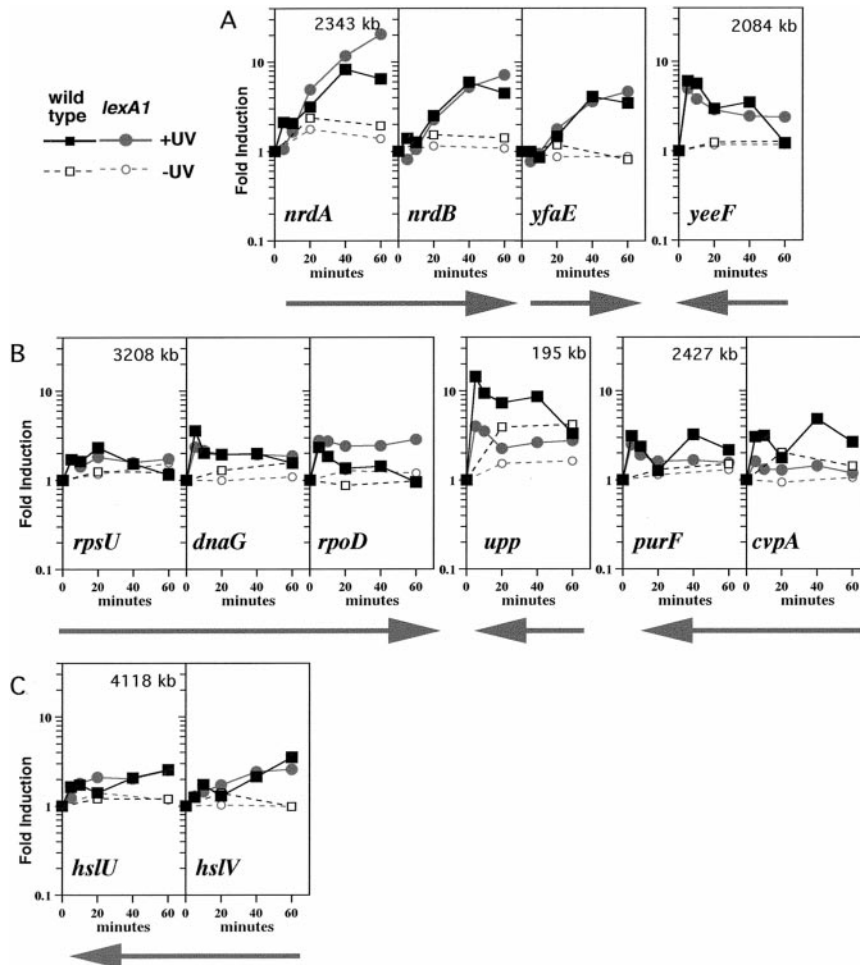


FIGURE 4.—Representation of genes displaying a LexA-independent transcriptional induction following UV irradiation. The change in transcript levels for the indicated gene is plotted as in Figure 2. (A) The largest LexA-independent inductions. (B) Typical profiles of genes with an early UV-dependent transcriptional induction. (C) Typical profiles of genes with a slow UV-dependent transcriptional induction. Solid squares, irradiated wild type; open squares, unirradiated wild type; solid circles, irradiated *lexA1*; open circles, unirradiated *lexA1*.

with other ATP-dependent helicases. The gene products of *ydeT*, *ydeS*, and *ydeR* share homology to other fimbrial proteins. *b1169ycgH* has homology with other ATP-binding subunits of transport systems. Both *ydeO* and *ydiW* have motifs that suggest they may function as transcriptional regulators. No significant homology between *ybiN*, *yqgC*, *yhiJL*, or *yifL* and any other characterized proteins has been reported.

**Genes induced independently of LexA following UV irradiation:** The time courses of the largest *lexA*-independent inductions are plotted in Figure 4A. Most striking is precisely how few *lexA*-independent changes occur following UV exposure. In general, *lexA*-independent inductions, with the exception of *nrdA*, *nrdB*, and *yeeF*, are in the range of twofold effects. Furthermore, many of these *lexA*-independent profiles appear to rise very rapidly (within the first 5 min) and then either subside or plateau. A large number of genes and regions were observed to be regulated in this manner and some generalizations are apparent from both Figure 1 and Table 2. Many proteins associated with the replication machinery are slightly induced following UV irradiation. Additionally, several genes associated with purine and pyrimidine metabolism seem to be upregulated in a similar manner. Although not dramatic, these results are partic-

ularly impressive considering that these nucleotide metabolism genes are often found in very small operons spaced throughout the genome. Other categories of genes that appeared to be upregulated included heat-shock or chaperone proteins as well as several of the genes involved in RNA metabolism. It is notable that nearly half of the genes that were upregulated have had little or no functional characterization.

**Loss of gene expression following UV irradiation:** Whereas several studies have focused upon the need to upregulate certain gene products following UV irradiation, it has remained relatively unexplored, yet very possible, that repression or even active degradation of some gene transcripts will also be an important factor in cellular recovery. The bacterial microarray offers an opportunity to address this very question. Indeed, repression was observed in a large number of genes following UV irradiation. However, our results do not allow us to determine whether the decrease in a given transcript represents diminished transcription or accelerated degradation in response to UV irradiation. Nevertheless, a large number of genes in the wild-type, but not in the *lexA1*, samples were reduced in their transcript levels at the 5-, 10-, and 20-min time points following irradiation. This observation may suggest that some inhibition of



**TABLE 2**  
**Genes with increased transcript levels following UV-irradiation**

Gene	WT (ave) <sup>b</sup>	lexAI (ave)	Wild type: minutes post irradiation <sup>c</sup>						lexAI: minutes post irradiation						Possible function		
			5	10	20	40	60	(No UV)	5	10	20	40	60	(No UV)			
<i>umuC<sup>a</sup></i>	20.6	0.974	5.22	20.59	15.58	39.36	22.74	1.11	0.9	0.95	0.91	0.95	0.97	1.04	1.04	0.94	PolV, SOS mutagenesis
<i>recN<sup>a</sup></i>	20.18	0.937	28.94	26.2	26.7	29.76	23.07	1.36	1.31	0.79	0.8	0.85	0.9	0.76	0.99	0.76	Protein used in DNA repair
<i>umuD<sup>a</sup></i>	17.36	1.046	5.13	12.42	28.56	27.07	8	1.18	0.69	0.94	1.01	1.11	1.01	1.03	1	0.95	SOS mutagenesis; forms complex with PolV
<i>recA<sup>a</sup></i>	10.08	1.251	6.16	6.28	9.26	8.49	4.57	0.6	0.78	1.09	0.95	0.93	1.04	1.12	0.8	0.84	Homologous strand pairing, DNA strand exchange
<i>dinB<sup>a</sup></i>	7.749	1.098	3.66	5.8	7.08	5.72	5.25	0.71	1.03	1.02	0.81	0.94	1.03	0.88	0.88	PolIV	
<i>uvrB<sup>a</sup></i>	4.142	1.496	3.16	4.42	4	5.03	4.62	0.94	1.11	1.15	1.11	1.35	1.63	1.68	0.98	0.87	DNA repair; excision nuclease subunit B
<i>uvrA<sup>a</sup></i>	3.846	1.167	1.96	1.79	1.68	1.37	1.18	0.39	0.44	1.07	1.01	0.88	0.85	0.83	0.74	0.85	DNA repair; excision nuclease subunit A
<i>polB<sup>a</sup></i>	3.552	1.043	4.54	4.6	3.07	3.76	3.3	1.06	1.11	1.16	1	1.1	1.07	1.2	1.15	0.97	Holliday junction helicase subunit B; branch migration
<i>polB<sup>a</sup></i>	3.2	1.098	1.46	1.7	1.61	1.65	2.14	0.52	0.55	0.91	0.76	0.73	0.82	2.19	0.91	1.06	DNA polymerase II
<i>ydjQ<sup>a</sup></i>	3.361	0.852	2.78	3.35	2.13	2.41	2.27	0.63	0.91	0.87	0.79	0.68	0.69	0.59	0.86	0.84	Putative excinuclease subunit
<i>uvrD<sup>a</sup></i>	2.505	1.127	1.25	1.02	1.22	1.47	1.49	0.47	0.56	1.1	1.03	1.17	1.2	1.33	1.09	0.98	DNA excision repair; helicase II
<i>ref</i>	1.792	1.675	1.22	1.1	0.99	0.98	1.31	0.59	0.66	2.01	1.65	1.11	0.98	0.91	0.86	0.73	ssDNA and dsDNA binding, ATP binding
<i>dnaN</i>	1.684	1.59	1.83	1.46	1.09	1.05	1.35	0.81	0.8	2.25	1.75	1.6	1.15	1.08	1.02	0.95	DNA polymerase III, $\beta$ -subunit
<i>dnaA</i>	1.627	1.633	2.45	1.32	1.59	1.2	1.37	0.84	1.11	2.57	2.07	1.72	1.35	1.27	1.03	1.17	DNA initiation of chromosome replication
<i>dnaC</i>	1.482	1.621	2.06	1.52	1.32	1.64	1.28	1.2	0.91	1.68	1.41	1.54	1.88	1.92	1.15	0.93	DNA replication initiation and chain elongation
<i>rep</i>	1.622	1.433	1.13	0.83	0.8	1.1	1.33	0.57	0.71	1.67	1.53	1.56	1.68	1.91	1.14	1.19	Rep helicase, chromosome replication
<i>ruvB<sup>a</sup></i>	1.931	0.889	3.19	2.96	2.66	2.78	2.22	1.42	1.44	1.01	1.01	1.02	0.86	1.09	1.12	0.98	Holliday junction helicase subunit A; branch migration
<i>ruvC</i>	1.375	1.452	1.01	0.76	0.89	0.81	0.62	0.5	0.69	1.58	1.51	1.55	1.49	1.64	1.16	0.98	Holliday junction nuclease; resolution of structures
<i>lexA<sup>a</sup></i>	4.8	0.914	3.21	3.07	3.08	2.77	2.51	0.61	0.61	0.84	0.6	0.67	0.6	0.49	0.78	0.62	Regulator for SOS(lexA) regulon
<i>dinF<sup>a</sup></i>	4.461	1.02	2.46	2.81	3.24	2.78	1.98	0.49	0.7	1.04	1	1.06	1.1	0.9	0.97	1.03	Damage-inducible protein I
<i>deaf</i>	1.479	2.819	1.31	0.65	0.85	0.64	0.58	0.5	0.59	2.11	1.35	1.62	1.7	2.1	0.65	0.61	Inducible ATP-independent RNA helicase
<i>rpoD</i>	1.71	2.131	2.33	1.84	1.36	1.43	0.95	0.87	0.98	2.78	2.73	2.41	2.43	2.86	1.28	1.2	RNA polymerase, sigma(70) factor
<i>hepA</i>	1.92	ND	1.84	1.82	1.77	2.16	1.53	0.89	1.01								Probable ATP-dependent RNA helicase
<i>fis<sup>a</sup></i>	2.294	1.366	3.02	2.19	1.94	2.01	1.22	0.83	0.98	1.63	1.43	1.56	1.44	1.52	1.05	1.17	DNA inversion factor; transcription factor
<i>subB<sup>a</sup></i>	2.081	1.555	1.94	1.33	1.35	1.6	0.96	0.69	0.69	2.38	2.07	2.25	2.56	2.71	1.44	1.64	Enhances synthesis of $\sigma$ 32 in mutant
<i>titK</i>	2.179	1.408	2.67	2.07	1.77	2.56	1.77	1.09	0.9	1.4	1.52	1.58	1.44	1.17	0.94	0.94	Putative transcriptional regulator
<i>ydcO<sup>a</sup></i>	2.412	1.009	1.64	3.37	10.33	2.98	1.58	1.58	1.65	1.13	1.11	1.19	1.23	1.32	1.18	1.19	Putative ARAC-type regulatory protein
<i>rph</i>	1.75	1.595	1.53	1.08	0.82	1.4	1.25	0.69	0.7	1.69	1.66	1.76	2.04	1.82	1.22	1.03	RNase PH
<i>srnB</i>	1.474	1.839	1.96	1.42	1.16	1.54	1.29	0.88	1.12	2.78	2.4	2.3	2.49	2.72	1.31	1.45	ATP-dependent RNA helicase
<i>soxS</i>	1.728	1.345	2.07	1.48	1.48	2.1	0.95	1.06	0.85	1.06	1.1	1.23	1.48	1.05	0.93	0.83	Regulation of superoxide response regulon
<i>rnpA</i>	1.696	1.329	2.32	1.47	1.24	1.34	1.05	0.81	0.94	1.45	1.28	1.24	1.29	1.45	0.94	1.08	RNase P component; processes tRNA, 4.5S RNA

(continued)

**TABLE 2**  
(Continued)

Gene	WT (ave) <sup>b</sup>	<i>lexA1</i> (ave)	Wild type: minutes post irradiation <sup>c</sup>						<i>lexA1</i> : minutes post irradiation						Possible function		
			5		10		20		40		60		20			60	
			(No)	(UV)	(No)	(UV)	(No)	(UV)	(No)	(UV)	(No)	(UV)	(No)	(UV)		(No)	(UV)
<i>nrdA</i>	2.051	5.046	2.11	2.06	3.15	8.28	6.5	2.37	1.94	1.06	1.66	4.91	11.74	20.49	1.77	1.39	Ribonucleoside reductase 1, $\alpha$ -subunit, B1
<i>grxA<sup>a</sup></i>	4.515	1.205	2.18	25.25	9.65	1.9	1.2	1.66	1.9	1.07	0.91	1	1.06	1.23	0.95	0.8	Glutaredoxin coenzyme for ribonucleotide reductase
<i>nrdB</i>	2.117	2.956	1.4	1.26	5.92	5.92	4.47	1.53	1.41	0.81	1.05	2.25	5.23	7.14	1.15	1.08	Ribonucleoside reductase 1, $\beta$ -subunit, B2
<i>upp</i>	2.126	1.922	14.45	9.43	7.34	8.62	3.32	3.93	4.19	4	3.53	2.26	2.63	2.76	1.53	1.63	Uracil phosphoribosyltransferase
<i>pyrF</i>	1.734	1.701	2.13	1.97	1.2	2.66	2.1	1.1	1.22	1.96	1.57	1.49	1.76	1.98	0.93	1.13	Orotidine-5'-phosphate decarboxylase
<i>tsx</i>	1.964	1.331	4.08	2.34	1.61	2.12	1.24	0.93	1.39	1.72	1.51	1.42	1.26	1.21	1.05	1.09	Nucleoside channel; phage T6 receptor and colicinK
<i>guaA</i>	1.833	1.447	2.04	1.89	1.08	2.09	1.47	0.91	0.96	1.72	1.89	1.5	1.6	1.61	1.05	1.25	GMP synthetase (glutamine-hydrolyzing)
<i>speB</i>	1.494	1.757	1.13	1.06	0.89	0.88	0.97	0.62	0.7	1.68	1.49	1.81	1.75	1.66	0.92	0.99	Agmatinase
<i>purF</i>	1.728	1.515	3.13	2.37	1.27	3.24	2.17	1.31	1.51	2.5	1.9	1.61	1.67	1.56	1.14	1.3	PRPP aminotransferase
<i>dpp</i>	1.601	1.602	2.9	1.95	1.96	2.19	2.65	1.41	1.5	2.06	1.62	1.8	1.78	1.75	1.29	0.96	Flavoprotein affecting DNA panthothenate metabolism
<i>pyrH</i>	1.602	1.56	2.36	1.33	1.13	1.8	1.23	0.85	1.11	1.71	1.53	1.53	1.72	1.56	1.01	1.08	Uridylate kinase
<i>nthA</i>	1.416	1.732	1.54	1.53	1.25	1.76	1.39	0.92	1.19	1.55	1.79	1.8	1.88	1.9	1	1.06	dATP pyrophosphohydrolase
<i>dut</i>	1.641	1.504	1.68	1.28	0.95	1.42	1.48	0.84	0.82	1.36	1.43	1.5	1.55	1.64	1.06	0.93	Deoxyuridinetriphosphatase
<i>gauB</i>	1.509	1.532	2.64	1.76	1.53	2.33	1.81	1.23	1.44	2.02	1.98	1.75	1.97	1.51	1.21	1.2	IMP dehydrogenase
<i>purB</i>	1.479	1.526	3.31	2.82	2.39	3.38	2.26	1.49	2.34	2.14	2.2	1.86	2.18	1.92	1.36	1.34	Adenylosuccinate lyase
<i>gpi</i>	1.666	1.319	2.85	2.01	2	1.9	1.03	1.09	1.26	1.67	1.51	1.36	1.48	1.4	1.01	1.24	Guanine-hypoxanthine phosphoribosyltransferase
<i>pyrD</i>	1.557	1.426	8.04	5.09	2.98	5.8	2.73	2.76	3.57	2.02	1.7	1.17	1.29	1.2	1.01	1.06	Dihydro-orotate dehydrogenase
<i>purH</i>	1.494	1.478	0.72	0.93	0.49	1.51	1.73	0.58	0.86	1.65	1.55	1.5	1.8	1.68	1.12	1.14	AICAR formyltransferase; IMP cyclohydrolase
<i>pyrE</i>	1.839	1.124	1.38	1.06	0.85	1.3	1.25	0.6	0.67	1.13	1.06	0.92	1.02	0.93	0.94	0.86	Orotate phosphoribosyltransferase
<i>murB</i>	1.457	1.438	3.18	2.01	2.35	2.68	2.6	1.63	1.89	1.2	1.17	1.25	1.33	1.09	0.87	0.81	UDP-N-acetylenolpyruvylglucosamine reductase
<i>cnk</i>	1.5	1.37	1.69	1.8	0.99	2.18	1.48	1.24	0.93	1.48	1.46	1.37	1.46	1.39	1.08	1.01	Cytidylate kinase
<i>hdsB</i>	1.623	1.225	0.82	1.11	0.66	1.85	1.32	0.7	0.72	1	1.15	0.81	1.05	0.95	0.85	0.77	CTP:UMP-3-deoxy-D-manno-octulosonate transferase
<i>argS</i>	1.708	1.645	2.51	2.8	1.59	3.36	2.55	1.67	1.33	1.86	1.7	1.46	1.83	1.91	1.13	1	Arginine tRNA synthetase
<i>prfC</i>	1.656	1.576	2.01	1.49	1.28	1.75	1.79	0.98	1.03	1.98	1.58	1.68	1.9	2	1.1	1.22	Peptide chain release factor RF-3
<i>aspS</i>	1.662	1.54	2.13	1.65	1.27	1.53	1.69	1.01	0.98	2.19	1.82	2.04	2.15	2.04	1.38	1.28	Aspartate tRNA synthetase
<i>speA</i>	1.485	1.715	1.62	1.49	1.1	1.26	1.25	0.87	0.94	2.12	2.24	1.76	1.87	1.7	1.03	1.23	Biosynthetic arginine decarboxylase
<i>prfB</i>	1.598	1.594	1.97	1.39	1.37	1.73	2.13	0.97	1.18	1.6	1.62	1.59	1.67	1.45	0.96	1.03	Peptide chain release factor RF-2
<i>fabZ</i>	1.698	1.492	3.63	2.7	2.71	2.94	1.82	1.64	1.61	1.51	1.46	1.5	1.66	1.59	1.04	1.03	(3R)-hydroxymyristol acyl carrier dehydratase
<i>igt</i>	1.682	1.507	2.2	1.65	1.64	1.96	1.59	1.07	1.08	1.9	1.84	1.86	1.9	1.69	1.2	1.24	tRNA-guanine transglycosylase
<i>yjeA</i>	1.759	1.298	1.85	1.63	1.14	1.4	0.93	0.95	0.63	1.58	1.39	1.45	1.45	1.43	1.05	1.2	Putative lysyl-tRNA synthetase
<i>gltX</i>	1.67	1.375	3.65	2.84	1.88	3.49	2.67	1.95	1.53	1.68	1.67	1.6	1.97	2.05	1.2	1.41	Glutamate tRNA synthetase, catalytic subunit
<i>queA</i>	1.613	1.381	1.22	0.84	0.83	1.05	0.9	0.61	0.59	1.8	1.5	1.55	1.58	1.82	1.14	1.25	Synthesis of queuine in tRNA
<i>ygfT<sup>n</sup></i>	1.747	1.123	1.16	1.15	1.9	1.73	0.96	0.89	0.69	1.16	1.05	1.14	1.34	1.15	1.01	1.07	Putative aminopeptidase

(continued)

**TABLE 2**  
(Continued)

Gene	WT (ave) <sup>b</sup>	<i>lexA1</i> (ave)	Wild type: minutes post irradiation <sup>c</sup>						<i>lexA1</i> : minutes post irradiation						Possible function		
			5		10		20		40		60		20			60	
			(No UV)	(UV)	(No UV)	(UV)	(No UV)	(UV)	(No UV)	(UV)	(No UV)	(UV)	(No UV)	(UV)			
<i>yggH<sup>a</sup></i>	5.823	1.086	4.16	20.67	10.37	2.07	1.29	1.91	1.13	1.08	1.12	1.17	1.58	1.04	1.2	Putative ATP-binding transport system component	
<i>gtaB<sup>a</sup></i>	5.737	0.902	1.65	4.64	3.84	3.82	0.74	0.49	1.05	1.08	1	1.1	0.91	1.11	1.17	PTS system, arbutin-like IIB component	
<i>yeeF</i>	3.086	2.815	6.08	5.67	3.5	1.22	1.25	1.27	4.98	3.8	2.87	2.44	2.38	1.17	1.17	Putative amino acid/amine transport protein	
<i>ybeW<sup>a</sup></i>	2.986	0.913	0.9	1.76	1.97	1.9	1.28	1.13	1.19	1.24	1.26	1.39	1.56	1.09	0.9	Putative dnaK protein	
<i>ihpB<sup>a</sup></i>	2.216	1.335	2.99	2.71	3.16	2.01	0.98	0.93	2.39	2.04	2.08	2.52	2.38	1.43	1.37	Heat-shock protein	
<i>conA</i>	1.805	1.63	1.88	1.37	1.15	2.21	2.01	0.98	0.93	2.04	2.08	2.52	2.38	1.43	1.37	Mg <sup>2+</sup> transport, system I	
<i>yhcC</i>	1.762	1.384	1.82	1.85	0.72	1.89	1.43	1.17	0.58	1.7	1.6	1.81	1.79	1.14	1.21	Putative transport	
<i>potB<sup>a</sup></i>	1.833	1.226	4.54	3.13	1.98	2.77	2.06	1.54	1.62	1.76	1.29	1.04	1.09	1.01	0.97	Probable serine transporter	
<i>hslU</i>	1.559	1.498	1.64	1.73	1.41	2.07	2.54	1.21	1.2	1.24	1.79	2.1	2.02	2.51	1.41	Spermidine/putrescine transport system permease	
<i>potC</i>	1.501	1.489	4.53	3.66	1.75	2.61	2.05	1.82	2.07	2.44	2.02	1.34	1.26	1.28	1.01	Heat-shock protein hslVU, ATPase subunit	
<i>uraA</i>	1.554	1.38	2.85	2.14	0.88	1.65	0.99	0.82	1.37	2.37	2.05	1.15	1.31	1.16	1.06	Spermidine/putrescine transport system permease	
<i>potD</i>	1.383	1.498	2.03	2.3	1.99	1.58	1.23	1.24	1.4	1.98	2.69	2.23	2.09	1.72	1.5	Uracyl transport	
<i>potA</i>	1.61	1.241	4.43	3.36	1.75	2.51	1.47	1.98	1.38	1.74	1.82	1.38	1.19	1.16	1.06	Spermidine/putrescine periplasmic transport protein	
<i>artP</i>	1.503	1.305	3.15	2.89	1.43	2.92	1.52	1.75	1.42	1.75	1.58	1.32	1.64	1.54	1.28	ATP-binding component of spermidine transport	
<i>salA<sup>a</sup></i>	9.561	1.108	13.74	11.7	13.17	11.55	9.36	1.05	1.44	0.85	0.92	0.76	0.9	0.89	0.67	Third arginine transport system, ATP-binding	
<i>ymjN<sup>a</sup></i>	7.57	1.034	0.09	3.35	7.12	0.54	0.39	1.1	1.03	1.14	1.1	0.93	1.15	1.15	0.9	Suppressor of lon; inhibits cell division	
<i>intE<sup>a</sup></i>	4.292	0.905	1.05	1.76	7.96	21.11	1.65	1.43	0.91	0.96	0.95	1.01	1.15	1.03	1.17	Orf, hypothetical protein	
<i>ymjF<sup>a</sup></i>	3.658	1.004	0.7	1.02	1.17	13.56	4.95	1.17	1.03	0.9	0.94	0.96	0.94	1	0.9	Prophage e14 integrase	
<i>ymjG<sup>a</sup></i>	2.514	0.996	0.75	1.31	1.05	4.5	6.28	1.1	1.11	0.98	1.04	1.03	1.12	1.01	1.03	Orf, hypothetical protein	
<i>ogrK<sup>a</sup></i>	2.191	1.242	2.98	4.7	6.89	3.23	1.59	1.69	1.85	1.12	1.11	1.14	2	1.09	0.94	Orf, hypothetical protein	
<i>ybcU<sup>a</sup></i>	1.854	1.004	9.35	5.6	3.68	5.15	2.97	2.61	3.16	1.16	1.05	1.02	1.03	1.01	1.02	Prophage P2 ogr protein	
<i>prfB</i>	2.159	ND	1.96	1.41	1.24	1.73	2.08	0.78						1.01	1.08	Bacteriophage λ Bor protein homolog	
<i>fabF</i>	1.788	2.249	3.05	2.23	2.53	2.73	1.8	1.38	1.38	1.8	1.89	1.97	2.24	2.95	1.02	0.91	3-oxoacyl-[acyl-carrier-protein] synthase II
<i>pphB</i>	2.743	1.189	2.18	3.18	8.5	1.29	1.24	1.3	1.09	1.11	1.02	1.01	1.07	1.08	0.94	0.84	Protein phosphatase 2
<i>yhdG</i>	2.087	1.744	1.83	1.59	1.24	1.66	0.88	0.68	0.7	2.38	2.01	2.13	1.93	1.84	1.18	1.18	Putative dehydrogenase
<i>hlyE</i>	1.894	ND	2.52	2.97	4.35	3.08	1.57	1.28	1.78								Hemolysin E
<i>ribH</i>	1.857	1.703	1.14	0.99	0.8	1.1	1.17	0.41	0.71	1.64	1.57	1.48	1.6	1.63	0.93	0.93	Riboflavin synthase, β chain
<i>gtdA</i>	1.678	1.558	1.14	0.87	0.78	0.93	1.02	0.52	0.61	1.39	1.42	1.65	1.46	1.44	0.83	1.06	Glucose-inhibited division; chromosome replication?
<i>prmA</i>	1.595	ND	1.29	1.18	1.04	1.31	1.12	0.63	0.86								
<i>capA</i>	1.791	1.368	3.05	3.15	1.77	4.85	2.67	2.03	1.43	1.61	1.31	1.29	1.44	1.19	0.94	1.06	Membrane protein required for colicin V production
<i>pabC</i>	1.555	ND	1.25	1.19	1.25	1.57	1.27	0.84	0.84								4-amino-4-deoxychoisinate lyase

(continued)



**TABLE 2**  
(Continued)

Gene	WT (ave) <sup>b</sup>	<i>lexAI</i> (ave)	Wild type: minutes post irradiation <sup>c</sup>						<i>lexAI</i> : minutes post irradiation						Possible function		
			5		10		20		40		60		20			60	
			(No UV)	(No UV)	(No UV)	(No UV)	(No UV)	(No UV)	(No UV)	(No UV)	(No UV)	(No UV)	(No UV)	(No UV)		(No UV)	
<i>pta</i>	1.66	1.444	1.72	1.73	1.14	2.57	2.26	1.26	1.01	1.47	1.73	1.76	1.99	2.11	1.2	1.31	Phosphotransacetylase
<i>nuoB</i>	1.567	1.492	2.11	2.46	1.23	3.12	2.48	1.51	1.4	2.17	1.48	1.53	1.48	1.32	1.09	1.05	NADH dehydrogenase I chain B
<i>gidB</i>	1.698	1.354	2.08	1.4	1.79	1.7	1.52	0.85	1.15	1.35	1.3	1.39	1.31	1.35	1	0.98	Glucose-inhibited division; chromosome replication?
<i>fldA</i>	1.491	1.474	5.95	4.41	4.5	5.5	3.64	3.29	3.15	1.8	1.51	1.68	2.11	2.22	1.17	1.36	Flavodoxin I
<i>plsX</i>	1.594	1.363	2.01	1.37	1.1	1.8	1.33	0.99	0.92	1.48	1.32	1.31	1.5	1.41	1.04	1.02	Glycerolphosphate auxotrophy in <i>plsB</i> background
<i>smgA<sup>e</sup></i>	1.95	0.984	2.15	2.05	2.6	1.96	1.67	1	1.14	1.03	1.01	0.94	0.86	0.93	0.94	1	Small membrane protein A
<i>smgB<sup>e</sup></i>	1.755	1.169	2.64	2.33	1.67	2.39	1.81	1.28	1.19	1.23	1.49	1.23	1.28	1.23	1.08	1.13	Small protein B
<i>emrB</i>	1.606	1.286	1.28	1.06	1.04	1.15	1.05	0.65	0.74	1.32	1.06	1.16	1.43	0.98	1.05	0.8	Multidrug resistance; putative translocase
<i>ackA</i>	1.541	1.342	3.41	2.91	2.97	3.27	2.62	1.92	2.02	1.23	1.34	1.25	1.37	1.52	0.99	1.01	Actate kinase
<i>fabG</i>	1.265	1.576	2.13	1.84	1.86	2.04	1.27	1.3	1.59	1.24	1.44	1.47	1.72	2.01	0.94	1.06	3-oxoacyl[acyl-carrier-protein] reductase
<i>sbp</i>	1.344	1.481	0.84	0.81	1.14	3.66	3.36	1.47	1.45	0.6	1.1	1.6	2.21	2.97	1.18	1.11	Periplasmic sulfate-binding protein
<i>dinD<sup>r</sup></i>	10.47	1.028	6.71	5.95	6.02	5.48	6.19	0.56	0.6	0.77	0.72	0.57	0.71	0.52	0.78	0.5	DNA-damage-inducible protein
<i>oraA<sup>e</sup></i>	9.002	1.087	4.83	5.99	6.2	8.43	3.13	0.67	0.6	1.09	1.05	1.08	1.04	1.04	0.97	0.98	Regulator, OraA protein
<i>yebG<sup>r</sup></i>	8.853	1.175	3.87	4.51	5.64	6.1	3.34	0.44	0.62	0.95	0.82	0.75	0.84	0.87	0.67	0.77	Orf, hypothetical protein
<i>yfaE</i>	2.18	2.69	1	0.86	1.47	4.09	3.48	1.19	0.81	0.77	0.94	1.78	3.61	4.67	0.87	0.88	Orf, hypothetical protein
<i>yigF<sup>n</sup></i>	3.813	1.057	1.84	15.18	3.02	2.03	1.19	1.22	1.09	0.98	0.97	1.08	1.06	1.02	0.94	0.93	Orf, hypothetical protein
<i>yigN<sup>n</sup></i>	3.969	0.866	2.11	2.38	2.13	3.13	4.14	0.66	0.74	0.79	0.82	0.86	0.76	0.71	0.89	0.95	Putative $\alpha$ -helix chain
<i>arhB<sup>r</sup></i>	3.749	0.996	2.29	12.29	5.32	2.58	1.61	1.23	1.34	1.08	1.01	0.95	0.94	0.8	0.97	0.95	Orf, hypothetical protein
<i>dinF<sup>n</sup></i>	3.621	1.029	2.1	1.98	1.26	1.81	1.54	0.58	0.38	1.02	1.06	0.87	0.84	0.84	0.95	0.85	DNA-damage-inducible protein F
<i>yafO<sup>r</sup></i>	3.486	1.031	3.73	4.97	3.51	3.35	2.83	0.8	1.31	0.98	0.96	0.86	0.96	1.11	1.02	0.87	Orf, hypothetical protein
<i>yegQ</i>	2.034	2.403	2.32	1.85	0.86	1.63	1.02	0.63	0.88	3.66	3.18	2.53	2.35	2.46	1.18	1.18	Orf, hypothetical protein
<i>yaaA</i>	2.182	ND	1.43	1.2	1.08	1.43	1.35	0.6	0.59								Putative enzyme
<i>yafN<sup>n</sup></i>	2.738	1.097	3	3.71	3.66	2.28	1.05	1.26	1.01	1.08	0.95			0.82	0.92	0.84	Orf, hypothetical protein
<i>yebF<sup>n</sup></i>	2.909	0.859	3.24	4.06	3.33	4.67	2.88	1.25	1.05	1.01	1	0.89	0.84	1	1.23	1.23	Orf, hypothetical protein
<i>yafP<sup>n</sup></i>	2.622	1.13	2.29	3.29	3.18	2.65	1.83	0.84	1.18	1.05	1	0.97	0.87	0.8	0.83	0.83	Orf, hypothetical protein
<i>ydiY<sup>n</sup></i>	2.197	1.539	4.65	2.39	1.85	2.1	1.37	1.07	1.18	2.86	1.59	1.4	1.28	1.18	1.02	1.14	Orf, hypothetical protein
<i>ydfM<sup>r</sup></i>	2.629	1.104	1.92	3.08	3.03	2.65	1.94	1.03	0.89	1.03	1.01	0.85	0.8	1	0.84	0.86	Orf, hypothetical protein
<i>yebC</i>	1.944	1.69	2.67	2.14	2.17	1.9	1.28	0.97	1.12	1.51	1.92	1.64	2.33	2.36	1.2	1.11	Orf, hypothetical protein
<i>yfgB</i>	2.064	1.555	2.05	1.29	1.08	1.33	1.06	0.6	0.72	1.56	1.41	1.38	1.44	1.48	0.96	0.91	Orf, hypothetical protein
<i>yfgO</i>	1.796	1.703	1.14	1.1	0.71	1.06	0.93	0.58	0.52	2.11	1.48	1.62	1.59	1.63	1.01	0.97	Putative enzyme
<i>yebE<sup>n</sup></i>	2.379	1.047	1.56	1.68	1.69	1.49	1.61	0.63	0.72	0.95	0.88	0.71	0.76	0.86	0.88	0.71	Orf, hypothetical protein
<i>ydgN</i>	1.931	1.454	1.95	1.05	0.96	0.99	1.23	0.54	0.74	1.85	1.35	1.31	1.26	1.21	0.95	0.97	Putative membrane protein
<i>yhgG</i>	1.774	1.544	2.14	1.76	1.3	2.28	1.3	1.01	0.97	2	1.58	1.7	1.95	1.84	1.16	1.19	Orf, hypothetical protein
<i>yhin<sup>n</sup></i>	2.072	1.222	2.9	2	1.72	2.8	2.13	1.15	1.08	1.31	1.16	1.1	1.3	1.24	1.02	0.98	Orf, hypothetical protein
<i>dcrB</i>	1.732	1.498	1.98	1.94	1.51	2.4	2.65	1.21	1.21	1.74	1.48	1.71	2.33	2.18	1.14	1.38	Orf, hypothetical protein

(continued)

**TABLE 2**  
(Continued)

Gene	WT (ave) <sup>b</sup>	<i>lexA1</i> (ave)	Wild type: minutes post irradiation <sup>c</sup>						<i>lexA1</i> : minutes post irradiation						Possible function		
			5		10		20		40		60		20			60	
			No (UV)	Ave (UV)	No (UV)	Ave (UV)	No (UV)	Ave (UV)	No (UV)	Ave (UV)	No (UV)	Ave (UV)	No (UV)	Ave (UV)		No (UV)	Ave (UV)
<i>ycaP<sup>a</sup></i>	2.028	1.183	1.79	2.21	1.93	1.44	1.45	0.87	0.87	1	0.92	1.03	0.96	0.94	0.8	0.84	Orf, hypothetical protein
<i>ycaH</i>	1.971	1.221	2.33	1.96	1.41	2.08	1.63	0.9	1.01	1.63	1.34	1.22	1.25	1.46	1.01	1.25	Orf, hypothetical protein
<i>yedM</i>	1.826	1.355	1.86	3.91	4.61	2.73	1.73	1.81	1.44	1.02	0.98	2.11	1.2	0.89	0.95	0.88	Orf, hypothetical protein
<i>ygwW</i>	1.582	ND	1.83	3.12	2.99	3.77	4.47	1.97	2.12								Orf, hypothetical protein
<i>yabU</i>	1.641	1.476	4.46	2.75	2.53	3.35	2.21	1.97	1.76	1.74	1.33	1.48	1.5	1.44	1.11	0.92	Putative nucleolar proteins
<i>yefB</i>	1.757	1.358	1.89	1.42	0.96	1.68	1.43	0.99	0.69	1.43	1.2	1.26	1.43	1.37	0.88	1.09	Orf, hypothetical protein
<i>yjiW<sup>a</sup></i>	2.255	0.855	2.49	2	1.8	1.71	1.47	0.7	0.98	0.97	1.02	0.98	1.07	0.81	1.15	1.12	Orf, hypothetical protein
<i>yifL<sup>a</sup></i>	2.185	0.907	1.55	1.43	1.63	1.65	1.17			0.68	0.93	0.9	0.71	0.64	0.87	0.93	Orf, hypothetical protein
<i>ybfE</i>	1.677	1.364	2.46	2.5	2.91	3.42	2.04	1.27	1.91	1.24	1.08	1.14	1.5	1.35	0.86	0.99	Orf, hypothetical protein
<i>yhbC</i>	1.276	1.754	1.78	0.99	1.09	0.77	0.76	0.82	0.87	1.41	1.06	1	0.95	0.93	0.62	0.6	Orf, hypothetical protein
<i>ydeT<sup>a</sup></i>	2.083	0.96	3.17	2.8	1.91	2.47	1.26	1.2	1.03	1.35	1.08	1.05	0.96	1.01	1.17	1.1	Putative outer membrane protein
<i>ygcM</i>	1.78	1.25	2.27	1.93	2.22	2.91	1.66	1.33	1.14	1.12	1.2	1.08	1.09	1.26	0.84	1	Putative 6-pyruvoyl tetrahydrobiopterin synthase
<i>yfiA</i>	1.548	1.467	2.71	1.48	1.32	1.95	1.48	1	1.31	1.43	1.48	1.57	1.6	1.88	1.09	1.08	Orf, hypothetical protein
<i>yehF</i>	1.613	1.394	2.34	1.96	1.53	2.07	1.7	1.1	1.28	1.84	1.77	1.85	1.87	1.87	1.27	1.37	Putative GTP-binding protein
<i>ygiR</i>	1.513	1.494	1.5	0.89	0.77	0.92	1.14	0.55	0.83	1.91	1.44	1.42	1.53	1.32	1.05	0.97	Orf, hypothetical protein
<b>IS5K</b>	1.696	1.292	1.82	1.44	1.52	1.75	1.23	0.93	0.9	1.05	1.18	1.07	1.15	1.04	0.83	0.87	Orf, hypothetical protein
<i>yfhE</i>	1.157	1.828	0.47	0.57	0.47	0.61	0.57	0.42	0.51	1.34	1.8	1.84	1.63	1.66	0.9	0.91	Orf, hypothetical protein
<i>yeeA</i>	1.753	1.229	2.78	2.71	2.63	2.53	1.84	1.47	1.38	1.04	1.14	1.02	0.99	0.85	0.87	0.77	Orf, hypothetical protein
<i>yaeS</i>	1.679	1.221	1.68	1.15	1.11	1.4	1	0.88	0.63	1.22	1.26	1.22	1.28	1.28	1.06	0.99	Orf, hypothetical protein
<i>yaaH</i>	1.706	1.193	1.53	0.95	0.84	0.9	0.77	0.46	0.71	1.1	1.15	1.08	0.9	0.99	0.93	0.82	Orf, hypothetical protein
<i>yefQ</i>	1.675	1.285	3.39	3.33	3.43	3.03	1.98	1.89	1.73	1.48	1.32	1.45	1.53	1.48	1.34	0.92	Orf, hypothetical protein
<b>b1228</b>	1.624	1.33	0.61	0.86	0.98	1.37	1.01	0.56	0.63	1.23	1.15	1.17	1.27	1.3	0.89	0.95	Orf, hypothetical protein
<i>ydfR<sup>a</sup></i>	1.884	1.055	1.76	2.17	1.58	1.68	1.71	0.85	1.04	1.03	0.84	0.94	0.91	0.92	0.95	0.81	Orf, hypothetical protein
<i>yfhH</i>	1.481	1.453	1.24	1.29	0.93	1.66	1.88	1	0.89	1.18	1.23	1.4	1.6	1.6	1.01	0.92	Orf, hypothetical protein
<i>ybfJ</i>	1.627	1.338	1.7	1.31	1.31	1.73	1.27	0.81	0.99	1.68	1.46	1.6	1.5	1.62	1.18	1.17	Putative enzyme
<i>yacL</i>	1.496	1.385	2.79	2.66	2.31	2.69	2.34	1.72	1.7	1.41	1.41	1.48	1.61	2.02	1.13	1.16	Orf, hypothetical protein
<i>yfgK</i>	1.395	1.499	1.1	0.99	0.98	1.12	1.18	0.74	0.8	1.13	1.33	1.47	1.5	1.69	0.91	0.99	Putative GTP-binding factor
<i>ydgQ</i>	1.449	1.415	1.67	1.28	1.14	1.04	0.74	0.74	0.88	1.53	1.17	1.17	1.08	1.03	0.88	0.81	Orf, hypothetical protein
<i>yieA</i>	1.512	1.345	1.45	1.19	1.01	1.21	0.96	0.66	0.88	1.18	1.45	1.02	1.13	1.24	0.89	0.9	Orf, hypothetical protein
<i>yiaD</i>	1.532	1.318	1.95	1.3	1.98	1.61	1.47	0.88	1.29	1.21	1.45	1.74	1.65	1.33	1.1	1.14	Putative outer membrane protein
<i>ygeQ</i>	1.424	1.423	2.43	1.57	1.73	1.54	1.34	1.04	1.09	1.18	1.06	1.15	1.09	1.21	0.74	0.86	Orf, hypothetical protein
<i>ycaP<sup>b</sup></i>	1.765	1.067	2.05	1.7	1.84	3.04	1.34	1.04	1.22	1.4	0.86	1.09	1.18	1.02	0.98	1.1	Orf, hypothetical protein
<i>yabO</i>	1.624	1.207	1.19	1.1	1.15	1.1	0.82	0.61	0.71	1.09	1.1	1.01	1.01	1.1	0.87	0.89	Orf, hypothetical protein
<i>yhdJ</i>	1.686	1.117	2.55	1.71	1.23	1.38	0.76	0.73	1.08	1.55	1.07	1.16	1.03	1.11	0.95	1.17	Putative methyltransferase

<sup>a</sup> Induced in a *lexA*-dependent manner.

<sup>b</sup> Ave represents the average transcript level in irradiated samples compared to that in unirradiated samples as described in MATERIALS AND METHODS.

<sup>c</sup> Values represent the fold change in transcript level at the time indicated compared to the beginning of the experiment as described in MATERIALS AND METHODS.

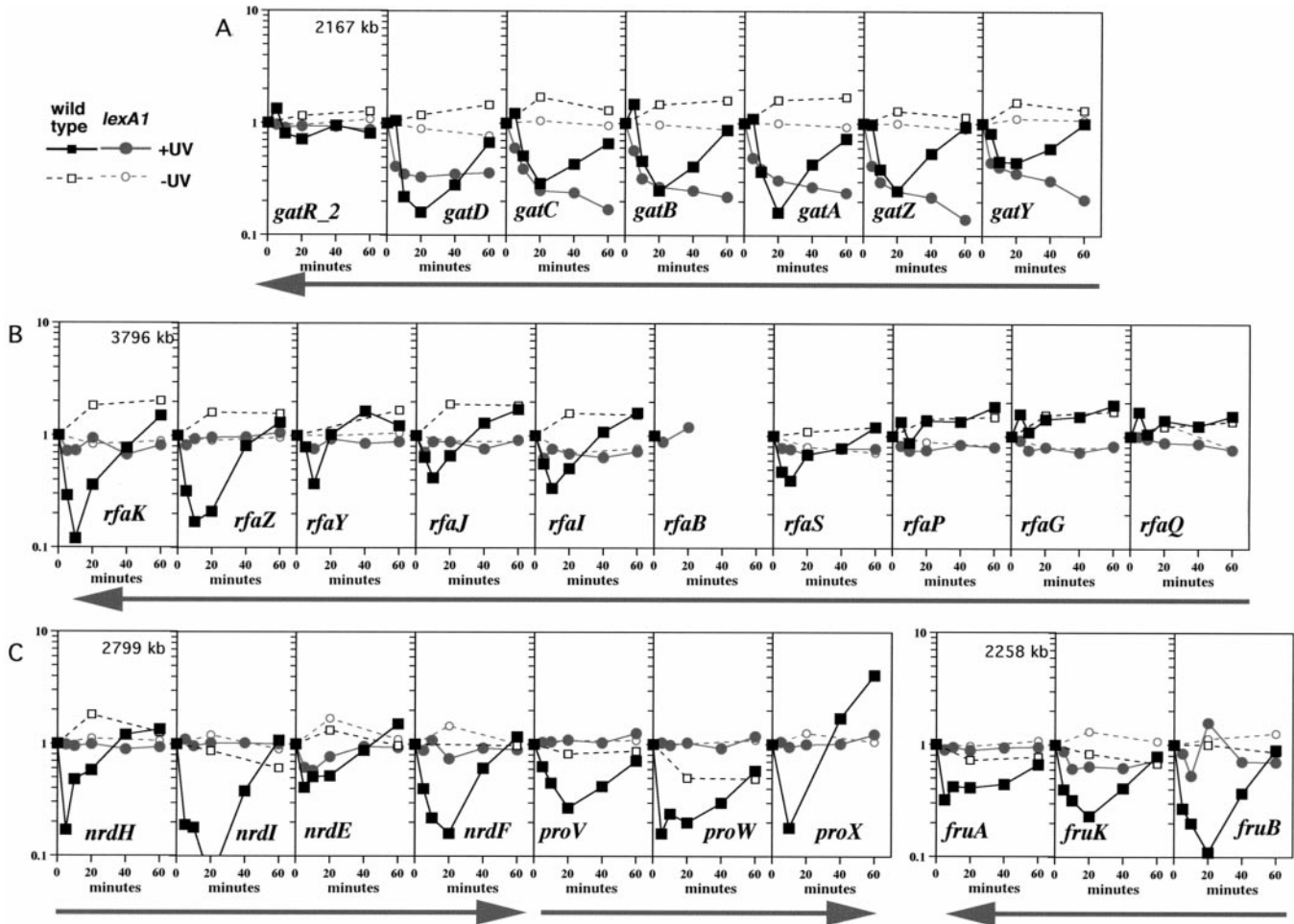


FIGURE 5.—Representation of genes that displayed a reduced level of transcripts following UV irradiation. The change in transcript levels for the indicated gene is plotted as in Figure 2. (A) A typical operon in which the transcript from both wild-type cells and *lexA1* mutants was observed to be reduced. (B) An operon in which the transcript from wild-type cells was observed to be severely reduced at the 5' end. (C) Three operons in which transcripts were reduced in wild-type cells throughout the operon.

transcription or degradation of transcripts occurs in a *lexA*-dependent manner, and it may point to a *lexA*-dependent mechanism for inhibition of transcription or enhanced degradation of these transcripts. Due to the small sample size of this experiment, we are inclined to interpret these findings cautiously. However, further investigation of these observations is clearly warranted.

The phenomenon of downregulation is also interesting to consider with respect to DNA repair. Actively transcribed genes in *E. coli* are repaired preferentially compared to nontranscribed genes (MELLON and HANAWALT 1989). When the *lac* operon is actively transcribed at the time of UV irradiation, repair of the transcribed strand occurs within 5 min after irradiation. While it is assumed that this response allows for the rapid transcriptional recovery of expressed genes, little is known about the actual inhibition or recovery of transcription for operons other than *lac* in *E. coli*.

Diminished transcript levels were clearly evident in some operons. Several different temporal profiles were observed (Figure 5). In some cases, exemplified by the

*gat* operon, the decrease in transcript levels following UV irradiation was observed in both wild-type and *lexA1* cells (Figure 5A). In the wild-type cells, the time at which transcripts recovered to preirradiation levels varied but generally occurred prior to 40 min postirradiation. Interestingly, whereas transcripts recovered to pretreatment levels in the wild-type cells, transcripts of several genes failed to recover in *lexA1* mutants within the period observed in these experiments, suggesting that *lexA*-regulated genes have an important role in this transcriptional recovery (Figure 5A).

The *gat* operon, controlling galactitol uptake and metabolism, is one of several operons involved in the metabolism of different carbon sources, which were found to have reduced transcript levels following UV irradiation. Other carbon metabolism operons whose transcript levels decreased include *fru*, *man*, *wwb*, *glg*, and *mal*. Our cultures were grown with glucose as the sole carbon source. It may be interesting to know how these metabolic pathways are regulated when cells are grown in the presence of their respective carbon sources.



**TABLE 3**  
**Operons with reduced transcript levels following UV-irradiation**

Operon	Location in kb and strand transcribed	WT <i>lexA1</i> (ave) <sup>a</sup>	Wild type: minutes postirradiation <sup>b</sup>												Possible function		
			<i>lexA1</i> : minutes postirradiation						<i>lexA1</i> : minutes postirradiation								
			5	10	20	40	60	UV	5	10	20	40	60	UV			
<i>araD</i>	65-	0.85	1.07	0.82	1.08	0.98	1.33	1.4	1.12	1.51	1.04	1.06	1.01	1.07	1.15	1	L-ribulose-5-phosphate 4-epimerase
<i>araA</i>		0.58	ND	0.48	0.38	0.84	0.79	0.93	1.22	0.93	1.08	0.98	0.99	0.96			L-arabinose isomerase
<i>araB</i>		0.65	1.05	0.63	0.71	0.24	1.04	1.16	1.14	1.19	0.97	1.05	1	1.05	1.13	1	L-ribulokinase
<i>modF</i>	791-	0.67	0.45	0.44	0.54	0.54	1.37	1.9	1.19	1.68	0.4	0.68	0.73	0.68	0.44	1.3	ATP-binding component of molybdate transport system
<i>ybiC</i>	835+	0.62	0.51	1.28	1.01	1.04	0.93	0.93	1.48	1.86	0.85	0.9	0.89	0.84	0.57	1.57	Putative dehydrogenase
<i>ybiK</i>	865+	0.83	0.93	0.74	0.84	1	1.52	2.22	1.44	1.59	0.49	1.06	1.38	1.42	1.13	1.2	Putative asparaginase
<i>yliA</i>		0.63	0.73	0.35	0.42	0.54	0.8	1.22	0.79	1.32	0.57	0.76	0.76	0.71	0.63	0.92	Putative ATP-binding component of a transport system
<i>yliB</i>		ND	0.79	0.36	0.36	0.43	0.65	0.88	0.85	0.89	0.3	0.46	0.7	0.59	0.46	0.95	Putative ATP-binding component of a transport system
<i>yliC</i>		0.62	0.56	0.47	0.34	0.42	0.55	0.79	0.8	0.91	0.71	0.85	0.85	0.82	0.71	0.93	Putative transport protein
<i>yliD</i>		0.6	0.83	0.47	0.34	0.42	0.55	0.79	0.8	0.91	0.71	0.85	0.85	0.82	0.71	0.93	Putative transport system permease protein
<i>minC</i>	1223-	0.74	0.61	0.58	0.32	0.44	0.63	0.78	0.85	0.63	0.46	0.49	0.65	0.61	0.52	0.89	Putative transport system permease protein
<i>mimD</i>		0.33	0.65	1.31	0.73	0.52	0.64	0.96	2.45	2.61	0.79	0.94	0.83	0.82	0.58	1.2	Cell division inhibitor, inhibits ftsZ ring formation
<i>mimE</i>		0.45	0.63	1.52	0.81	0.6	0.74	0.78	2.1	1.84	0.8	0.93	0.88	0.72	0.54	1.37	Cell division inhibitor, a membrane ATPase, activates minC
<i>treA</i>	1244-	0.33	0.94	1.03	0.59	0.46	0.58	0.64	1.87	2.17	0.96	0.98	1.02	1.1	0.84	1.05	Cell division topological specificity factor
<i>ygcC</i>	1246-	0.48	0.7	2.29	1.15	1.27	1	1.09	3	2.7	0.99	0.89	0.88	0.8	0.84	1.27	Trehalase, periplasmic
<i>ygsS</i>		0.49	0.54	0.65	0.65	0.71	0.88	1.23	1.39	1.96	0.54	0.59	0.58	0.56	0.4	0.99	Putative PTS system enzyme I
<i>ygtT</i>		0.69	0.67	0.77	0.52	0.35	0.58	0.88	0.88	0.92	0.77	0.74	0.72	0.7	0.67	1.12	Putative dihydroxyacetone kinase
<i>ygdL</i>	1500+	0.8	ND	0.99	0.59	0.62	0.77	1.01	0.86	1.12	0.81	0.8	1.14	0.79	0.7	1.12	Putative dihydroxyacetone kinase
<i>yddU</i>	1561-	ND	0.78	0.57	1.97	1.23	1.43	0.9	0.77	2.19	2.05	0.9	0.85	0.72	0.43	0.31	Putative dihydroxyacetone kinase
<i>yddV</i>		0.59	0.57	0.71	0.57	0.73	0.56	0.73	2.29	2.61	0.85	0.97	0.92	0.73	0.88	1.07	Orf, hypothetical protein
<i>pqqL</i>	1570-	ND	0.71	0.72	0.79	0.77	0.91	0.88	1.24	1.05	0.72	0.79	0.77	0.91	0.88	1.24	Putative enzyme
<i>yddB</i>		0.38	0.66	0.93	0.6	0.64	1.18	1.25	2.25	2.54	0.68	0.83	0.84	1.19	1.01	1.7	Orf, hypothetical protein
<i>yddA</i>		0.6	0.87	0.73	0.5	0.84	1.2	1.19	1.3	1.67	0.96	1.1	1.08	1.21	1.14	1.3	Putative ATP-binding component of a transport system
<i>yeaG</i>	1864+	0.5	0.63	1.41	1.32	1.46	1.1	0.96	2.86	2.18	0.88	0.93	1.07	0.72	0.58	1.49	Putative peptidase
<i>yeaH</i>	1866+	0.5	0.62	0.61	0.62	0.95	0.58	0.52	1.2	1.44	0.69	0.95	0.84	0.65	0.58	1.21	Orf, hypothetical protein
<i>manX</i>	1900+	0.58	0.44	1.16	0.51	0.39	0.54	0.75	1.01	1.31	0.71	0.67	0.46	0.38	0.3	1.28	Orf, hypothetical protein
<i>manY</i>		0.55	0.55	1.18	0.72	0.33	0.61	0.74	1.3	1.31	0.82	0.64	0.53	0.48	0.42	1.07	PTS enzyme IIB, mannose-specific
<i>manZ</i>		0.73	0.6	1.23	0.65	0.48	0.55	0.7	0.92	1.05	0.82	0.74	0.57	0.49	0.42	1.06	PTS enzyme IIC, mannose-specific
																	PTS enzyme IID, mannose-specific

(continued)

**TABLE 3**  
(Continued)

Operon	Location in kb and strand transcribed	WT <i>lexA1</i> (ave) <sup>b</sup>	Wild type: minutes post irradiation <sup>b</sup>												Possible function			
			minutes post irradiation <sup>b</sup>						<i>lexA1</i> : minutes post irradiation									
			5	10	20	40	60	UV	5	10	20	40	60	UV				
IS51	2099-	0.98	1.16	1.07	0.7	0.56	0.76	0.76	0.56	1.01	1.15	1.1	1.17	1.31	1.28	1.01	1.06	IS5 transposase
<i>wbbL</i>		0.49	0.89	0.61	0.25	0.41	1.12	1.25	1.27	1.7	0.73	0.77	0.76	0.86	0.96	0.92	0.91	Putative creatinase
<i>wbbK</i>		0.47	0.66	0.49	0.24	0.39	1.3	1.6	1.53	1.91	0.27	0.26	0.29	0.45	0.83	0.69	0.58	Putative glucose transferase
<i>wbbJ</i>		0.55	0.74	0.77	0.53	0.79	1.41	1.65	1.64	2.12	0.48	0.54	0.56	0.71	0.71	0.79	0.83	Putative O-acetyl transferase
<i>wbbI</i>		0.41	0.93	0.52	0.5	0.9	1.37	1.31	2	2.46	0.61	0.69	0.79	0.87	0.93	0.87	0.8	Putative GalF transferase
<i>rjc</i>		0.53	1.14	0.37	0.3	0.59	1.05	0.95	1.14	1.3	0.6	0.59	0.71	0.75	1.04	0.68	0.61	o-Antigen polymerase
<i>glf</i>		ND	0.88								0.62	0.64	0.62	0.7	0.88	0.79	0.78	UDP-galactopyranose mutase
<i>galR_2</i>	2069-	0.76	0.9	1.33	0.8	0.71	0.94	0.8	1.15	1.26	0.96	0.9	0.93	0.92	0.86	0.95	1.07	Split galactitol utilization operon repressor, fragment 2
<i>galD</i>		0.36	0.43	1.05	0.22	0.16	0.28	0.67	1.18	1.46	0.41	0.35	0.33	0.35	0.36	0.89	0.77	Galactitol-1-phosphate dehydrogenase
<i>galC</i>		0.41	0.33	1.22	0.51	0.29	0.43	0.66	1.71	1.3	0.6	0.39	0.25	0.24	0.17	1.05	0.95	PTS system galactitol-specific enzyme IIC
<i>galB</i>		0.45	0.35	1.48	0.46	0.25	0.41	0.87	1.47	1.6	0.57	0.32	0.27	0.25	0.22	0.97	0.88	Galactitol-specific enzyme IIB phosphotransferase system
<i>galA</i>		0.34	0.35	1.1	0.37	0.16	0.43	0.73	1.61	1.71	0.49	0.39	0.31	0.27	0.24	1	0.93	Galactitol-specific enzyme IIA phosphotransferase system
<i>galZ</i>		0.51	0.28	0.98	0.39	0.25	0.54	0.93	1.29	1.14	0.42	0.3	0.25	0.22	0.14	1	0.89	Putative tagatose 6-phosphate kinase 1
<i>galY</i>		0.46	0.32	0.82	0.46	0.45	0.6	1	1.55	1.32	0.45	0.41	0.36	0.31	0.21	1.11	1.08	Tagatose-bisphosphate aldolase 1
<i>fruA</i>	2260-	0.6	0.9	0.32	0.42	0.41	0.44	0.66	0.72	0.77	0.89	0.94	0.88	0.93	0.94	0.96	1.07	PTS system, fructose-specific transport protein
<i>fruK</i>		0.57	0.58	0.4	0.32	0.23	0.41	0.79	0.83	0.68	0.87	0.61	0.64	0.62	0.73	1.31	1.07	Fructose-1-phosphate kinase
<i>fruB</i>		0.4	0.73	0.27	0.2	0.11	0.37	0.9	1	0.86	0.84	0.53	1.57	0.71	0.7	1.13	1.26	PTS system, fructose-specific IIA/fpr component
<i>yfbE</i>	2363+	0.49	0.53	1.66	1.17	0.85	0.88	0.97	2.91	1.61	0.85	0.79	0.85	0.8	0.8	1.44	1.62	Putative enzyme
<i>malP</i>	3545-	0.5	0.67	0.97	0.71	0.75	0.61	1.04	1.73	1.55	0.76	0.72	0.92	0.78	0.64	1.28	1.01	Maltodextrin phosphorylase
<i>malQ</i>		0.56	0.84	0.69	0.32	0.56	0.39	0.47	0.87	0.87	0.98	0.93	1.02	0.95	0.79	1.19	1.04	4- $\alpha$ -glucanotransferase (amylomaltase)
<i>glpD</i>	3560+	0.56	ND	0.22	0.37	0.4	0.3	0.61	0.66	0.69								sn-glycerol-3-phosphate dehydrogenase (aerobic)
<i>glgP</i>	3561-	0.62	0.55	0.49	0.3	0.5	0.51	0.66	0.68	0.9	0.44	0.5	0.74	0.68	0.64	1.15	1.04	Glycogen phosphorylase
<i>glgA</i>		0.39	0.59	0.3	0.26	0.43	0.37	0.49	0.74	1.16	0.46	0.5	0.75	0.79	0.66	1.1	1.06	Glycogen synthase
<i>glgC</i>		0.63	0.63	0.57	0.53	0.5	0.64	0.87	1.01	0.96	0.5	0.66	0.8	0.79	0.62	1.05	1.1	Glycose-1-phosphate adenyltransferase
<i>glgX</i>		0.64	0.54	0.33	0.48	0.5	0.61	0.86	0.94	0.81	0.36	0.49	0.68	0.57	0.47	0.99	0.91	A glycosyl hydrolase, debranching enzyme
<i>glgB</i>		0.71	0.62	0.72	0.67	0.45	0.8	0.99	0.94	1.11	0.56	0.72	0.84	0.7	0.64	1.17	1.05	1,4- $\alpha$ -glucan branching enzyme
<i>nlpA</i>	3836-	0.61	0.43	1.47	1.11	1.05	1.9	2.4	2.6	2.62	0.49	0.62	0.62	0.52	0.46	1.25	1.3	Lipoprotein-28
<i>aceB</i>	4213+	0.67	0.55	1.48	1.23	0.78	1.24	1.95	2.37	1.61	0.77	0.79	0.71	0.55	0.43	1.35	1.02	Malate synthase A
<i>aceA</i>		1	0.64	1.82	0.56	0.57	0.49	0.97	0.81	0.96	0.74	0.67	0.58	0.53	0.35	1.01	0.77	Isocitrate lyase
<i>aceK</i>		0.46	0.44	0.59	0.46	0.37	0.38	0.79	1.14	1.11	0.42	0.44	0.36	0.3	0.24	0.9	0.71	Isocitrate dehydrogenase kinase/phosphatase

<sup>a</sup> Ave represents the average transcript level in irradiated samples compared to that in unirradiated samples as described in MATERIALS AND METHODS.

<sup>b</sup> Values represent the fold change in transcript level at the time indicated compared to the beginning of the experiment as described in MATERIALS AND METHODS.

A second form of repression profile is exemplified by the *rfa* operon, which encodes gene products involved in lipid synthesis in the membrane. The *rfa* operon displayed a loss of transcript following UV irradiation in wild-type cells, but not in the *lexA1* mutant. The loss of transcript was more severe at the 5' end of the operon, which may at least partially reflect the fact that most RNA degradation in *E. coli* is believed to occur 3'-5' (Figure 5B).

A third pattern of transcript reduction can be seen in *nrdHIEFproVWX* and *fruAKB* operons shown in Figure 5C. In these cases, loss of the wild-type transcripts occurred uniformly throughout the operons.

The most severely reduced transcripts from operons for which we obtained signal are listed in Table 3. Of interest to note is the repression of operons such as *minCDE* following UV irradiation. These genes are required to induce and regulate septum formation prior to cell division and it is interesting to consider the loss of these transcripts with respect to the induction of other genes that regulate cell division such as *sulA* and *ftsK*. Also worth noting is that *minC*, which is severely repressed, actually contains a LexA-like box, which is predicted to be a very good LexA-binding sequence (FERNANDEZ DE HENESTROSA *et al.* 2000). Dramatic reductions in transcript levels at predicted LexA boxes were also observed for *rfaJ* and *metE* (Figure 5B and Table 3). While it is impossible from these experiments to determine if the repression is directly due to a LexA regulation, we did observe cases where functional LexA boxes appeared to result in inhibition of gene expression following UV irradiation. The loss of transcription from *araDAB* following UV irradiation seems likely to be due to the upregulation of *polB*, which is located proximal to this operon (Figure 1 and Table 3). Thus, although LexA serves as a negative regulator for *polB*, it essentially behaves as a positive regulator of *araDAB* expression.

The study we have presented should serve as a starting point for follow-up projects. It provides some generalization with respect to the role of LexA in the regulation (up and possibly down) of genes in response to one type of environmental stress. Surprisingly, it shows us that, in the absence of LexA regulation, there are no other major responses to UV irradiation at the level of transcriptional regulation. It remains to be determined whether the minor changes are significant in terms of the overall stress response. The values and raw data for any or all of the genes can be retrieved either via the web locations listed in MATERIALS AND METHODS or upon request.

We thank Nicholas Cozzarelli and David Botstein for initiating and supporting experiments with bacterial microarrays. We thank C. Richmond, Y. Wei, and F. Blattner for their help in preparing a library of *E. coli* ORFs. J.C. is currently being supported by a European Molecular Biology Organization fellowship ALTF 265-1999. A.K. is a PMMB/NSF Postdoctoral Fellow (grant DMS-9406348). B.P. was

supported by the National Institutes of Health (NIH), Molecular Biophysics training grant GM08295-11. The work was supported by NIH grants to Nicholas R. Cozzarelli and David Botstein. P.O.B. was supported by the Howard Hughes Medical Institute and NIH grant HG00983. P.O.B. is an associate investigator at the Howard Hughes Medical Institute. Research by P.C.H. is supported by an Outstanding Investigator grant CA44349 from the National Cancer Institute.

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Communicating editor: P. L. FOSTER