Sigma38 (rpoS) RNA Polymerase Promoter Engagement via −10 Region Nucleotides

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Band shift assays using DNA probes that mimic closed and open complexes were used to explore the determinants of promoter recognition by sigma38 (rpoS) RNA polymerase. Duplex recognition was found to be much weaker than that observed in sigma70 promoter usage. However, binding to fork junction probes, which attempt to mimic melted DNA, was very strong. This binding occurs via the non-template strand with the identity of the two conserved junction nucleotides (−12T and −11A) being of paramount importance. A modified promoter consensus sequence identified these two nucleotides as among only four (underlined) that are highly conserved, and all four were in the −10 region (CTAACcT from −13 to −7). The remaining two nucleotides were shown to have different roles, −13C in preventing recognition by the heterologous sigma70 polymerase and −7T in directing enzyme isomerization. These −10 region nucleotides appear to have their primary function prior to full melting because probes that had a melted start site were relatively insensitive to substitution at these positions. These results suggest the sigma38 mechanism differs from the sigma70 mechanism, and this difference likely contributes to selective use of sigma38 under conditions that exist during stationary phase.

The alternative sigma factor, sigma38 (rpoS), is the principle regulator of stress responses in Escherichia coli. The sigma38 regulon is very large and controls from 50 to 100 genes (1). Subsets of these genes are induced during starvation for various nutrients and in response to various stresses such as the accumulation of reactive oxygen species and changes in pH and osmolarity. The highest activity of sigma38 occurs during stationary phase when these and others stresses are actively assaulting the cell. Many factors contribute to this activity, especially the increased stability of sigma38 (2).

Subsets of genes in this regulon are often needed at lower levels under conditions when sigma38 is not very active. Such genes can contain multiple promoters that are recognized by sigma38 and sigma70 (3–5) or have a single promoter that can be transcribed by both holoenzymes, although not necessarily to the same level (6, 7). The two holoenzymes can also be affected by common activator proteins, although again not necessarily to the same extent (8, 9). Thus the promoters of genes transcribed by sigma38 appear to have varying degrees of ability to be recognized by sigma70. In principle, the extent to which a promoter will be used by each sigma would be set by its core promoter sequence and by the upstream activator sites.

It is not known how promoter sequences specify the extent of transcription by each type of holoenzyme. Sigma38 and sigma70 have very similar amino acid sequences. The regions of sigma70 that recognize promoter DNA, conserved region 4.2 (recognizes the −35 element) and conserved regions 2.3–2.5 (recognize the −10 element) are greater than 70% similar to those of sigma38 (10). The two sigmas recognize similar but not identical DNA sequences near −10, but sigma38 promoters have less conservation near −35 (11). Footprinting has demonstrated that promoter contacts made by the two types of holoenzymes are similar as is the extent of DNA melting in the open transcription complex (9, 12). Moreover, it has been shown that sigma70 and sigma38 holoenzymes can transcribe many of the same genes in vitro (13). It seems that there is substantial overlap in promoter recognition by the two sigmas and that this contributes to cell physiology by allowing maximum flexibility in expression of genes required under different physiological conditions.

The understanding of differential use of promoters by the two sigmas has centered on three areas: variation in core promoter sequence, variation in macromolecular activators, and the influence of global non-protein effectors such as DNA supercoiling (14) or small molecules (15, 16). All three are believed to contribute to differential transcription. In this report we focus on the effects of core promoter sequence. Most recent work suggests that the sigma70 −35 consensus sequence may not be used by sigma38 (17, 18). Therefore we focus on how sigma38 uses the −10 region sequence, which is similar but not identical to that of sigma70. The questions address whether differences in mechanism occur because of the differences in promoter sequence and how these may contribute to selective transcription.

The most recent sigma38 consensus CTATACT (from −13 to −7) differs from the sigma70 consensus ATATAAG in the two underlined positions (11). The five common nucleotides include the three (−12T, −11A, and −7T) that are of dominant importance for sigma70 recognition (19, 20). There have been a few mutagenic studies of the sigma38 sequences, and these support the importance of −13C, −12T, and −7T in maximizing the level of sigma38 transcription (21, 22). Besides this information, little is known about the role of these and other nucleotides in sigma38-dependent transcription. Recently, we have used band shift analysis to investigate the role of the −10 region in sigma70-dependent transcription (23). Because the −10 region is the locus of DNA opening such studies included probes that mimicked the double strand: single strand fork...
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**EXPERIMENTAL PROCEDURES**

Sigma38 was overexpressed from pETF (13), and sigma70 was overexpressed from pQE30-rpoD (25). Both proteins were purified with the same protocol as described previously (26). *E. coli* RNA polymerase is a commercial product from Epicenter Technologies. All oligonucleotides were synthesized by Operon Technologies and were gel purified and prepared as described previously (24).

Electrophoretic mobility shift assays (EMSA)

Electrophoretic mobility shift assays (EMSA) were conducted as follows: 20 nM core RNA polymerase and either 60 nM sigma38 or 60 nM sigma70 were added to a 9-μl reaction mixture with 1× buffer B (50 mM Tris-HCl at pH 7.9, 200 mM potassium glutamate, 3 mM MgCl₂, 0.1 mM EDTA, 1 mM dithiothreitol, 100 μg/ml bovine serum albumin, 6 ng/ml heparin). This mixture was incubated at room temperature for 10 min to allow holoenzyme formation. The reaction was then placed on ice for 5 min before the addition of 1 μl of the annealed DNA probe. Reactions were further incubated for 20 min on ice, and for heparin challenge experiments, 0.5 μl of 1 mg/ml heparin was added for an additional 10 min. All samples were run on 7% polyacrylamide gels at 300 V with cold 1× Tris borate-EDTA with the apparatus packed in ice. After electrophoresis, the radioactive bands were visualized and quantified by PhosphoImager analysis. Each experiment was conducted at least four times, and the average percentage of binding was taken. Sigma70 experiments were also repeated with 100 μM KCl instead of 200 mM potassium glutamate. The results were similar.

**RESULTS**

**An Updated Consensus for Sigma38 Promoters**

We updated the consensus promoter sequence for sigma38 (see Supplemental Table I) by applying strict criteria and adding many new promoters (Fig. 1). A problem with such alignments is that many promoters can be transcribed in vitro by both sigma70 and sigma38. But it is difficult to know if this reflects sigma38-dependent transcription or just overlapping recognition by very similar sigmas. Earlier alignments included promoters that could be transcribed in vitro by sigma38 RNA polymerase but were not dependent on sigma38 in vivo, such as the lacUV5 and trp promoters (11). The current alignment contains 41 promoters that show an in vivo dependence on sigma38 and that have had their start sites mapped. Undoubtedly, some promoters have been missed, but the stricter criteria make the consensus likely to be more sigma38-specific.

The new alignment shows a similar but not identical consensus to that obtained before (11). The CTATACT –10 sequence identified previously differs from the new majority sequence (CTACACT from –13 to –7) only at position –10. Positions –10, –9, and –8 are not as well conserved as in prior alignments and are not better conserved than several other positions with 40–60% conservation. In fact, in viewing the promoter as a whole (Fig. 1), four nucleotides stand out in terms of conservation (70–95%), –13C, –12T, –11A, and –7T. Recall that the latter three are the same ones that are most important (and most strongly conserved) in sigma70 promoters (19, 20, 27). There are also AT-rich patches near positions –19 and –28.

One aim of this work was to learn the role of –10 region nucleotides so we used a hybrid promoter that was sigma38-specific and contained the majority sequence from –13 downstream. The upstream segment was from the fic promoter, which is preferentially recognized by sigma38 in vitro (13, 18) but does not contain the majority –10 region sequence. The hybrid promoter, called the fic consensus (fic con), was fic from –14 upstream and the majority from –13 downstream (Fig. 2A) with the majority defined by the most common nucleotide in the Fig. 1 alignment. Probes for EMSA experiments used this as the parent sequence with the downstream segment in either duplex or single strand form.

**Weak and Nonselective Recognition of Duplex Probes**

Initial EMSA experiments used several duplex DNA probes (Fig. 2A) to compare recognition by sigma38 and sigma70 holoenzymes. The experiments were conducted at 4 °C to minimize the open conformation of the DNA and protein so as to mimic closed complex formation. Addition of heparin abolished all binding to these duplex probes confirming the lack of open complex formation (data not shown). Fig. 2B (left) shows that the sigma38 holoenzyme can form weak complexes with most of these duplex probes (see the band marked with an arrow and note its absence in the lane with core only). However, the level of binding was very low, never surpassing 3% of the available probe. There was no discrimination between sigma38 and sigma70 promoters because the latter were bound equivalently (rightmost lane). The low level of binding required the presence of the –10 element as the truncated probe 12/12 showed no binding. We infer that sigma38 holoenzyme binds DNA with a –10 region but does so weakly and with a low degree of discrimination for its own promoter.

In the converse experiment, the sigma70 holoenzyme was tested on the same probes (Fig. 2B, right). Sigma70 also bound the sigma38-dependent fic probes weakly. Not surprisingly, sigma70 was able to discriminate its own promoter because it bound it strongly (see the rightmost lane). In so far as these results mimic closed complex formation, it appears that the sigma38 promoter duplex DNA is recognized weakly and indiscriminately by both types of holoenzyme.

**Tight and Selective Binding to Fork Junction Probes**

DNA melting within the –10 region follows duplex recognition in the
transcription initiation pathway. The DNA in this open state can be partially mimicked by using fork junction probes, which are duplex upstream and single-stranded from the non-template strand, binding became very strong (greater than 50% binding of the 7/12 probe in Fig. 3A). This is compara-
tible with that seen previously for sigma70 holoenzyme binding to homologous non-template single-strand fork structures. The 7/12 probe was bound at ~54% by the sigma38 holoenzyme and at 6% by the sigma70 holoenzyme. B, heparin challenge after holoenzyme incubation reduced sigma38 holoenzyme binding to ~21% and sigma70 holoenzyme binding to 3%.

Removal of nucleotide -7 decreased binding approximately 5-fold (compare probes 7/12 and 8/12). Further truncation of the single-stranded tail abolished binding. This pattern persisted in a heparin challenge protocol (Fig. 4, right). Sigma70 holoenzyme could not bind any probe other than 7/12 (data not shown). It appears that the sigma38 holoenzyme requires the entirety of the 10 region in fork junction form for maximal binding. Similar results have been obtained on a homologous sigma70 system (24).

The removal of nucleotides may have reduced binding either because recognition determinants had been eliminated or because the length of the tail had been reduced. To determine the importance of nucleotide identity in the context of the tightly bound fork junction structures, single substitutions were made. The results suggest that both nucleotide identity and the length of the single strand tail are determinants of binding.

A “G scan” of the majority sequence CTACACT (-10 element) was conducted in which the nucleotides were individually changed to guanosine. For most positions, guanosine was the nucleotide least represented in the promoter alignment (Fig. 1). The results are quite striking for sigma38 holoenzyme binding (Fig. 5A). Changing either -12T or -11A to G abolished binding by sigma38 RNA polymerase (Fig. 5A, top). Substitutions at the other positions had lesser consequences varying from no change to 3-fold reductions in binding. We also substituted the AT blocks near positions -19 and -28 with GC blocks, but these had little or no effect (not shown).

The data demonstrate that the identity of the base pairs that
constitute the junction between double strand and single strand DNA are of paramount importance for tight binding. Substitutions in the single strand tail beyond these junction nucleotides had only modest effects. Because the effect of deleting these same tail nucleotides was to abolish binding (Fig. 3) it seems that the length of the tail is also important in providing a sequence-nonspecific component for binding.

When this same series of probes was tested for sigma70 holoenzyme binding, some aspects of the results were very surprising. There were two important differences, one of which is relevant to the issue of how sigma38 promoters can be recognized in a selective manner. Substituting for the is important in preventing recognition by the sigma70 holoenzyme. The presence of −13G did not cure the defects caused by changes in these three positions (Fig. 6B), confirming that these conserved positions dominate DNA binding by sigma70 at a sigma38 promoter.

Changes Occur When the DNA Single Strand Overlaps the Start Site—When full open complexes form, the melted region includes not only the −10 region but also encompasses the transcription start site. To attempt to mimic this situation, EMSA was done with fork junction probes in which the single strand extended to +1. As in the previous experiments, a G scan of nucleotides between −13 and −1 was conducted. The results are displayed in Fig. 7A for the sigma38 holoenzyme.

These results were surprising in that all substitutions appeared to be well tolerated. No substitution reduced binding more than 2-fold. Similar results were obtained with the double substitutions described above (not shown). This was quite different from the results using probes with the shorter non-template strand. In that case, substitution at either −12 or −11 essentially abolished binding. It appears that when the melted region encompasses the start site, the identity of the fork junction nucleotides assumes a lesser importance in directing tight binding.

For the sigma70 holoenzyme, the presence of single-stranded start site sequences increased binding to the fic con promoter. Indeed this probe was bound comparably to the two holoenzymes. However, sigma70 was more dependent upon nucleotide identity because substitutions at positions −11 and −7 strongly reduced binding (Fig. 7B). These same substitutions abolished binding in the experiments that used shorter probes (Fig. 5B). It appears that the two holoenzymes have different sequence requirements in maintaining binding stability in the context of probes containing the fully melted non-template strand. Only the sigma70 holoenzyme relied on elements of the −10 consensus. This effect was probably because it was a sigma38 promoter that was being bound; when a strong sigma70 promoter containing a −35 region is assayed, the −10 region is not very important in analogous assays (23).

In addition, the effect of −13C, which contributed strongly to selectivity on the shorter probe, was detectable but less substantial in the context of this fully extended probe. In the new context, selectivity was more closely related to positions −11 and −7 (compare the two panels of Fig. 7), which are conserved positions at both sigma70 and sigma38 promoters. In the context of this melted sigma38 promoter, recognition by sigma70 but not by sigma38 required that these positions match the consensus.

The isomerization of the enzyme into its heparin-resistant form accompanies full open complex formation. In the preceding experiments, heparin challenges were not very revealing, but this was not the case when a fully opened probe mimic was assayed (Fig. 8). In this assay, the identity of nucleotides −12, −11, and −7 became important for both sigma38 and sigma70.
holoenzyme binding. That is, although these positions were not important for maintaining tight binding by the sigma38 holoenzyme, they played a role in directing the required conversion of the enzyme into its final heparin-resistant conformation. The highly conserved $3T$ seems to play a critical role for isomerization of the enzyme but only in the presence of the transcription start site.

**DISCUSSION**

We have used band shift assays to explore how sigma38 recognizes and uses promoter DNA. The results imply that sigma38 polymerase uses a distinct DNA recognition pathway. Below we discuss this pathway in the context of its relationship to sigma70 promoter recognition and in terms of its relevance to the physiology of sigma factor utilization.

**Promoter Selectivity and Recognition**—We derived a modified consensus sequence for sigma38 promoters by focusing solely on promoters known to be dependent upon sigma38 in *vivo*. Two features of this analysis are noteworthy. First, the data do not show any conserved nucleotides corresponding to a $-35$ region as some prior studies have suggested (30). Second, within the downstream majority sequence CTACACT (from $-13$ to $-7$) only four nucleotides (underlined) appear to be strongly conserved. Thus in terms of core promoter sequence, the data suggest that typical sigma38 promoters do not have an upstream element and have a downstream element with fairly low sequence complexity.

A very recent report (29) has identified a similar, but not identical, sigma38 consensus sequence (CTATACT) by comparing a subset of the promoters aligned here. The four highly conserved nucleotides are identified in both alignments. T or G at position $-14$, a position not identified as conserved in this alignment, also seemed to play a moderate role in sigma38 transcription.

Studies of sigma70 promoter recognition have made it clear that promoters lacking a $-35$ region cannot be transcribed easily in the absence of activator proteins (31). The data in the current study confirm this at the level of DNA duplex recognition. The lack of an upstream element probably accounts for a significant component of selectivity by denying sigma70 the use of sigma38 promoters. In fact, when a $-35$ region is added to a $-10$ con $1/1$ probe, duplex binding by sigma70 reaches 20%$^2$.

The current data indicate that the downstream element also

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$^2$ S. J. Lee and J. D. Gralla, unpublished results.
contributes to promoter selectivity but primarily through a single nucleotide position. Of the four strongly conserved nucleotides only \(13\) differs from the sigma70 consensus element. The presence of this nucleotide was shown to strongly inhibit binding by the sigma70 holoenzyme (Fig. 5). A further analysis (not shown) indicates that \(C\) is the least common nucleotide at \(13\) in a collection of sigma70 promoters. We believe that the lack of a \(35\) region and the presence of a \(T\) at position \(13\) prevent the recognition of sigma38 promoters by the sigma70 holoenzyme.

This leaves the critical issue of how sigma38 recognizes its own promoters. The current data indicate that the identity of each of the four conserved nucleotides is important for recognition. However, this does not explain how a small region could be sufficient to direct efficient recognition especially when sigma70 recognition requires many more conserved nucleotides. We have mutated various blocks in the upstream region and have found no evidence of an upstream recognition determinant (not shown). This suggests that in most cases sigma38 recognition needs assistance by various factors that work in conjunction with the CTACACT \(-10\) region.

The greater need for ancillary factors in sigma38 promoter recognition is consistent with results from band shift assays. Sigma38 promoter probes containing duplex DNA were bound at extraordinarily low levels. This maximal 3% binding is in strong contrast to the 25% probe binding by sigma70 holoenzyme to the duplex \(PR\) probe. Indeed, sigma38 cannot recognize \(PR\) (or \(lac\) UV5; not shown) efficiently even though they contain a \(35\) element. However, the addition of a \(35\) element to the fic con 1/1 probe increases sigma38 binding to 20%. Such use of a \(35\) element by sigma38 can occur (21), but promoters with a \(35\) element have decreased holoenzyme selectivity (21, 30). Overall, it appears that sigma38, under certain contexts, can utilize a \(35\) element, but its absence from the majority of sigma38 promoters is related to the need to prevent cross-recognition of sigma38 promoters that could deplete sigma70 RNA polymerase during exponential growth.

Upon induction, activators and effectors could enhance the poor duplex binding of sigma38. In fact, an earlier study suggested low superhelical density DNA preferentially increased sigma38 closed complex formation (14), and high potassium glutamate concentrations also seem to stimulate closed complex binding.3

3 R. Yang and J. D. Gralla, unpublished data.
The observation that sigma38 recognizes elements are largely a subset of sigma70 elements may be related to the need for occasional cross-transcription. Examples include osmE and dps, both of which are sigma38 promoters that also need to support sigma70 transcription (6, 7). These promoters contain either a −35 region or a −10 extended region upstream from a −10 region that contains a −13C and in the case of osmE, a poorly conserved −10 region. This would allow some level of use by both sigma70 and sigma38.

Role of the −10 Region in Open Complex Formation by Sigma38 Holoenzyme—There are only four highly conserved nucleotides in the current sigma38 consensus sequence (underlined in the CTACACT majority sequence), and they appear to play three different roles. These contributions were tested in a context (probe 7/12) that attempted to mimic the state of DNA when the −10 region has become melted. As discussed above, the −13C plays a major role in preventing sigma70 binding and a minor role in sigma38 binding. The −12T/−11A nucleotides play a major role in sigma38 binding affinity. This occurs when the −11A is melted to form a double-strand/single-strand fork junction. Substitution for either of these nucleotides essentially eliminates binding in this context. Positions −10 to −7 also increase binding affinity when they are single-stranded, but this effect is largely sequence-nonspecific; substitutions have minor effects on binding, whereas deletions decrease binding more strongly. The data show that these contributions are made by the non-template single strand.

When the melted non-template strand extends to encompass the transcription start site (probe 1/12), the role of the −10 region changes. First, each conserved nucleotide can be substituted, and the effect on binding is minimal (Fig. 7). Strong interactions by core polymerase are known to occur over the −6 to +1 segment (33). Apparently, these interactions relieve the strong dependence on the fork junction nucleotides. This suggests that if melting occurs at the fork junction and spreads downstream (34), the melting pathway may first use the −12−11 nucleotides to create a tight fork junction complex and subsequent downstream melting to fully stabilize the open complex.

Recent studies have shown that conserved nucleotides can play a role in converting the sigma70 RNA polymerase to the heparin-resistant state associated with functional open complexes (23). This also appears to be the case for sigma38, and it is here that the conserved −7T plays a role. When the probe encompasses the start site, substitution for either −12T, −11A, or −7T nearly abolishes binding in a heparin challenge assay. In the G scan of Fig. 7, these substitutions have little effect on binding without heparin. Because the DNA is already melted, the effect is associated with a change in the state of polymerase rather than the actual melting of the DNA. We infer that the −10 region assists in the isomerization of the enzyme to its heparin-resistant form. But this appears to occur primarily after the DNA has been fully melted to encompass the start site, which differs from the situation with the sigma70 holoenzyme.

Overall, the sigma38 open complex pathway is dependent on the conserved nucleotides to create a fork junction, probably to start melting, and to isomerize the holoenzyme prior to initiation. In contrast to sigma70, it is designed to do this with lesser contributions to duplex DNA recognition from the core promoter elements. A future challenge will be to learn how physiological effectors enter this pathway to promote these steps. These effectors seem to be designed to minimize assistance at the melting step, which the current data suggests favors sigma38 over sigma70. However, this needs to be established directly, as does the means by which duplex recognition is achieved, before the switch from sigma70 to sigma38 usage is understood.

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