Name of the Sequence. Ovine lipoprotein lipase cDNA.

Genus and Species. Ovis aries.

Origin of Clones. Because almost the whole 3′-untranslated region (3′ UTR) was found to be lacking from the ovine lipoprotein lipase (LPL) cDNA sequence (1,656 bp including the 5′ UTR, the coding sequence, and the first 44 bp of the 3′ UTR) published by Edwards et al. (1993), we have undertaken to characterize this region, with the aim to provide a full-length LPL cDNA. First, a 779-bp LPL 3′ UTR cDNA was obtained from ovine adipose tissue by rapid amplification of cDNA end (3′ RACE) using the following primers: forward 5′-GTATAGTGGCCAAATAGCACA-3′ and reverse 5′-TCAAGCTTCTGCAGGATCCTTTTTTTTTTTTTTTTTTTTTTTTTT-3′. The reverse primer was also used to prime reverse transcription. The RACE products were cloned into Smal-digested and dephosphorylated pGEM-4Z. The LPL recombinant plasmids were screened by PCR performed directly on colonies. Second, the sequence of the fragment lacking between the previous 779-bp fragment and the fragment sequenced by Edwards et al. (1993) was amplified by PCR with the forward 5′-CAAGCTTCTGCAGGATCCTTTTTTTTTTTTTTTTTTTTTTTTTT-3′ and reverse 5′-GATTTCCAGTAATAGCCTCTG-3′ primers. Sequencing reactions were carried out on both strands, using an ABI 373A automated DNA sequencer and the accompanying software (PE Applied Biosystems, Courtaboeuf, France).

Comparison with Related Sequences. The full-length sequence now known (Figure 1) is 72, 74, 82, and 95% identical to the human (Wion et al., 1987), rat (Brault et al., 1992), pig (Harbitz et al., 1992), and partial cow (Senda et al., 1987) sequences, respectively. Similar homologies are obtained for comparisons of the different 3′ UTR.

Sequence Data. The full-length ovine LPL cDNA sequence is 3,529 bp long with a 178-bp 5′ UTR, followed by a 1,434-bp open reading frame (Edwards et al., 1993) and a 1,917-bp 3′ UTR (present study). This

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Figure 1. Full-length sequence of the ovine lipoprotein lipase cDNA. The 178-bp 5′-untranslated region and the 1,434-bp (bold type) coding sequence were published by Edwards et al. (1993) (Accession number X68308). We report here the 1,917-bp of the 3′-untranslated region. (Accession number AF228667). The polyadenylation signals are underlined.
1,917-bp 3′UTR sequence has revealed three polyadenylation signals giving rise to three differently sized LPL mRNA in ovine adipose tissue (Bonnet et al., 1998). This is supported by the detection of 3′RACE PCR products (779 and 382 bp) all containing a polyA tail, differing in their lengths due to the use of different polyadenylation signals. Two of these polyadenylation signals (giving rise to the 3.4- and 3.8-kb LPL mRNA) are preferentially used. The third one (1.7 kb) seems to be poorly used because it is barely detectable in Northern blot analyses (Bonnet et al., 1998).

**Comments.** Lipoprotein lipase is the rate-limiting enzyme in triglyceride-rich lipoprotein catabolism, providing triglyceride-derived fatty acids to adipose tissue and muscle for storage and/or energy production. The regulation of the LPL gene is complex and may occur at the level of transcription, translation, and/or post-translational processing (Enerbäck and Gimble, 1993). The proximal 3′UTR of the LPL mRNA was shown to be involved in this complex regulation through its first 24 nucleotides, which contain the protein-binding domain responsible for the translation inhibition by epinephrine in human adipose tissue (Ranganathan et al., 1997). Elsewhere, preliminary studies in humans (Ranganathan et al., 1995), guinea pigs (Enerbäck et al., 1988), and sheep (Bonnet et al., 2000) have shown that the expression pattern of the differently sized LPL mRNA is different in adipose tissue and cardiac muscle. This suggests that the frequency at which polyadenylation sites are used differs between these tissues, thus reflecting a putative tissue-specific pretranslational regulation of LPL gene expression. Moreover, starvation/refeeding studies in rodents have disclosed a regulation of total LPL mRNA levels in adipose tissue and oxidative muscles. Nevertheless, it was not known whether changes in nutritional status affect the tissue-specific pattern of LPL gene expression. Characterization of the 3′UTR sequence, by which LPL mRNA differ, allowed us to quantify each of their forms by real-time RT-PCR. We have shown a lack of preferential regulation within tissue by nutritional status for one of these two mRNA species (Bonnet et al., 2000).

**Literature Cited**


**Key Words:** Complementary cDNA, Lipoprotein Lipase, Sheep