Staphylococcal enterotoxins have long been recognized as being responsible for staphylococcal food poisoning. Recently, attention has centered on their role as superantigens in exacerbating sepsis (4, 9, 12). Of the known enterotoxins, staphylococcal enterotoxin A (SEA) is one of the most commonly reported in human isolates (5, 9, 10). The human pathogen Staphylococcus aureus is the species most frequently associated with SEA production, although other species of Staphylococcus occasionally carry the SEA gene (3). One strain of S. caprae has been shown immunologically to produce SEA (20); however, this was not confirmed with genetic analysis. This is the first report of a S. caprae strain simultaneously carrying both the SEA gene and the staphylococcal enterotoxin-like toxin type P (SElP) gene. SEIP was recently described in a study reporting the whole-genome sequencing of S. aureus strain N315 (11). The toxin produces an emetic response in Suncus murinus (the house musk shrew), but the emetic activity of SEIP has not yet been tested on a primate model (13).

S. caprae is considered to be primarily an animal pathogen. However, it has been documented as a human pathogen responsible for bone and joint infections (1, 8, 17, 18) and sepsis/bacteremia (14, 15, 19). Most human infections involving S. caprae have been nosocomial, but community-acquired infections have also been reported (15). The isolate reported here was obtained from an inanimate surface at the Texas Department of Transportation offices in San Angelo, TX. The identity of the isolate was determined by using an API Staph kit (bioMeirux Inc., Durham, NC), internal transcribed spacer PCR (2), and sequence analysis of the bacterial rpoB gene (6). Results of a modified Ouchterlony double-diffusion test (7) suggested that this isolate was either producing SEA or showing a cross-reaction to it. Because this was unexpected, PCR amplification using previously described SEA primers (16) was performed to ascertain the presence of the SEA gene.

S. aureus ATCC 43300 and S. aureus ATCC 13565 were used as the negative and positive controls for SEA, respectively. The enterotoxin PCR product was successfully amplified in both the S. caprae isolate and the S. aureus ATCC 13565 control. Because a recent study reported that all SEA gene-specific primers described in the literature can also be used for successful amplification of the SEIP gene (16), multiple clones from each PCR were sequenced to address the possibility that both genes might be present in this strain of S. caprae. A total of 439 base pairs of the SEA gene was sequenced for 17 S. caprae clones and 9 S. aureus ATCC 13565 clones. Cross-referencing with the GenBank database showed the greatest nucleotide identity (99%) to the S. aureus enterotoxin A gene (GenBank accession number M18970) for 9 of the 17 S. caprae clones and all 9 of the S. aureus ATCC 13565 clones. However, the remaining eight S. caprae clones showed greatest nucleotide identity (98%) to the gene now known as the SEIP gene (GenBank accession number BA000018) (13).

The average divergence between the nucleotide sequence of the SEA gene and the SEIP gene obtained from S. caprae was 14.4%. The average divergence between the S. caprae SEA and SEIP amino acid translations was 17.5%.

In addition to being the first report of PCR amplification of both SEA and SEIP genes in a single strain of S. caprae, this study also reports the first time that the SEIP gene has been amplified in a species of Staphylococcus other than S. aureus. Whole-genome sequencing would confirm our results, but we are unaware of any such project involving S. caprae. These results warrant further investigations of enterotoxins in S. caprae.

Nucleotide sequence accession numbers. The GenBank accession numbers for the sequences obtained in this study are DQ641635 to DQ641670.

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