INTRODUCTION

Hypotrichosis, the complete or partial absence of normal hair, has been identified as a heritable trait in many mammalian species including mice, rats, cats, dogs, guinea pigs, and primates (reviewed by Hedrich 1994). Interest in hypotrichotic mutants often extends beyond the hair and skin anomaly, since many of the known hypotrichotic mutations generate pleiotropic effects, including athymia (Festing *et al.* 1978, Pantelouris 1973), immunodeficiency and autoimmune disease (Shultz 1988), defective cellular immunity (Morrissey *et al.* 1980), susceptibility to viral leukemia (Meier *et al.* 1969), and progressive nephrosis (Marit *et al.* 1995). In addition, rodent models that lack a full coat often offer a distinct advantage in studies involving wound healing (Burgess *et al.* 1990), transdermal migration of chemicals (Peck *et al.* 1988), percutaneous drug absorption (Auclair *et al.* 1991, Bradley *et al.* 1990 Chanez *et al.* 1989, Morimoto *et al.* 1992, Rougier *et al.* 1987, Rougier *et al.* 1990, Twist and Zatz 1989), and skin pharmacology (Bouclier *et al.* 1987, Rommain *et al.* 1991).

In related work, Guerriero (1929a) holds the view that all differentiated elements derive from a single stem cell. Snyder's well known studies on the sow (1923) and the human (1924) do not support this view. Similarly, Lancaster *et al.* (1946) refute this hypothesis.

Ultimately, the identification of the wild-type *shn* gene will allow molecular access to one more component essential to the normal development of mammalian skin and hair. With the gene in hand, and systems available for the manipulation and transfer of engineered genes in both whole animals and in tissue culture (see Li and Ho