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Information for Contributors appears on pages 112–114 of the 6 January 1995 issue. Editorial correspondence, including requests for permission to reprint and reprint orders, should be sent to 1333 H Street, NW, Washington, DC 20005.
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LETTERS

Wagnerian Genetics

The recent report of an "Abnormal fear response and aggressive behavior in mutant mice deficient for α-calcium calmodulin kinase II" by Chong Chen *et al.* (14 Oct., p. 291) provides what may be an unusual insight into the presumably inherited deficiency manifested by a certain Siegfried Volsung. While his entire pedigree has long been open to speculation, it is asserted that he was the offspring of the consanguineous mating between brother (Siegmund Volsung) and sister (Sieglinde Neidung, née Volsung), who were separated at birth, only to reunite in early adulthood (R. Wagner, *Die Walküre*, Act I). Although Mendelian genetics was awaiting rediscovery at the time this kindred became the subject of a lengthy report (*Der Ring des Nibelungen*, 1876), such laws of inheritance would predict that Siegfried was significantly at risk for genetic disorders. Indeed, it is a wonder that the only phenotypic evidence of consanguineous parentage was a *complete lack of fear*. In a manner somewhat comparable to the α-CaMKII–deficient mice described by Chen *et al.*, Volsung was also disposed to remarkable acts of defensive aggression and risk-taking behavior [for example, Siegfried versus Fafner (*Siegfried*, Act II)].

While genetic counseling was not generally available to the community in which he lived, Siegfried is unlikely to have heeded prudent advice since, in typical fashion, he fearlessly won the affection of his aunt Brunnhilde (*Siegfried*, Act III). Because the murine machismo reported by Chen *et al.* clearly demonstrates a dominant inheritance pattern, one must scrutinize the behavioral phenotypes of Siegfried's parents for evidence of intermediate forms of fearlessness. And, in fact, usual precaution is not a feature of their daring escape from Sieglinde's oppressive domestic trappings while at the same time singing constantly at great volume in the middle of the night (*Die Walküre*, Act I). The first and second filial offspring of the inevitable proband-aunt (Siegfried-Brunnhilde) mating may have provided valuable insight into the penetrance and mode of inheritance in this unusual disorder; however this will never be known because a complicated family dispute ended in not only Siegfried's death but the immolation of all known inhabitants of the region (*Götterdämmerung*, Act III, final scene).

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Response: We appreciate that Vogel brings to our attention the fascinating story of Siegfried Volsung, as depicted in Wagner's opera *Der Ring des Nibelungen*. Our previous work has shown that an autosomal dominant mutation in the α-CaMKII gene is associated with a phenotype of increased defensive aggression and a lack of fear. In contrast, the neuropsychiatric condition exhibited by Siegfried, whose parents are brother and sister, seems to be derived from an autosomal recessive mutation. Thus, it is not clear at all whether there is any genetic parallelism between the α-CaMKII heterozygous knockout mouse and the man. There are, of course, other possible interpretations. For example, Siegfried may have carried a sporadic mutation in the α-CaMKII gene; or one of his parents may have had heterozygous or homozygous mutation in the α-CaMKII gene. Siegfried's father, Siegmund, appears to share similar traits. As Bernard Shaw has characterized [*The Perfect Wagnerite: A Commentary on the Ring of the Nibelungs* (Constable, London, 1956)], "The boy Siegfried inherits . . . all his father's hardihood. The fear against which Siegmund set his face like flint, and the woe which he wore down, are unknown to the son. . . ." If Siegfried's mother, Sieglinde, is normal, both the father and the son may have had the heterozygous mutation. In this case, the genetic parallelism may be justifiable.

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Teaching Engineers and Scientists

The Policy Forum by Mary Lowe Good and Neal F. Lane "Producing the finest scientists and engineers for the 21st century" (4 Nov., p. 741) contains little that is either new or provocative. It reads like a sermon based on the gospel according to the Office of Science and Technology Policy and the National Science Foundation,