In medical genetics, the penetrance of a disease-causing mutation is the proportion of individuals with the mutation who exhibit clinical symptoms. Men who carry a *BRCA1* or *BRCA2* mutation are at increased risk for male breast cancer, although the lifetime risk for this is low. Familial cases usually have *BRCA2* rather than *BRCA1* mutations. Easton *et al.* (1997) studied breast cancer with linkage to 13q12-q13, in two families. They estimated that the cumulative risk of breast cancer in female carriers was 59.8% by age 50 years (95% confidence interval (CI) 25.9 to 78.5%), and 79.5% by age 70 years (95% CI 28.9 to 97.5%) and that in male carriers was estimated to be 6.3% by age 70 years (95% CI 1.4 to 25.6%). Risch et al. (2001) demonstrated that the estimated penetrance *BRCA2* mutations was greater for males (53%) than for females (38%). Per the Breast Cancer Linkage Consortium (1999), amongst *BRCA2* carriers, men are at increased risk of pancreatic cancer, melanoma, and an aggressive form of prostate cancer. The modes of inheritance for *BRCA2* mutations: (i) Autosomal dominant inheritance (HPO, OMIM, Orphanet), and (ii) Multifactorial inheritance (HPO, Orphanet).

	High penetrance	Moderate penetrance	Low penetrance
Genes	*BRCA2, BRCA1*	*CHEK2, PALB2*	2q35, 6q25.1 (*ESR1*), 10q21.2, 11q13.3, 12p11.22, 14q24 (*RAD51L1*) and 16q12.1 (*TOX3*)
Population frequency	<0.1%	MAF 1%	MAF >10%
Cancer risk (odds ratio)	>10.0	>2.0	0.76–1.57
Functional effect	Direct effect of mutation	Direct effect of variant	Direct effect of variant; linkage disequilibrium with causal variants
Strategy for identification	Sequencing of candidate genes	Sequencing of candidate genes	Case–control studies; genome-wide association study (GWAS)

Table 1: Classes of male breast cancer genetic susceptibility and comparison of their different features. Source: https://academic.oup.com/annonc/article/24/suppl_8/ viii75/238093 MAF: minor allele frequency.

Management of MBC usually consists of radical mastectomy; and subsequently, adjuvant radiotherapy and hormonal therapy with or without chemotherapy. Maxwell *et al.* 2017 demonstrated that complete loss of BRCA1/2 function is a requirement for efficacy of platinum agents and PARP inhibitors. Unlike patients carrying homozygous *BRCA 1/2* mutations, germline heterozygous mutations in these two genes confers significantly less sensitivity to these therapeutics. Sakai et al. (2008) showed that secondary intragenic mutations in *BRCA2* could restore the wildtype *BRCA2* reading frame, thereby conferring acquired resistance to cisplatin.