Entrez PubMed
Overview
Help | FAQ
Tutorial
New/Noteworthy
E-Utilities

PubMed Services
Journals Database
MeSH Database
Single Citation Matcher
Batch Citation Matcher
Clinical Queries
Special Queries
LinkOut
My NCBI (Cubby)

Related Resources
Order Documents
NLM Mobile
NLM Catalog
NLM Gateway
TOXNET
Consumer Health
Clinical Alerts
ClinicalTrials.gov
PubMed Central

1: Mol Cell Endocrinol. 1997 Jun 20;130(1-2):43-51. Related Articles, Links

Functional and structural analysis of R607Q and R608K androgen receptor substitutions associated with male breast cancer.

Poujol N, Lobaccaro JM, Chiche L, Lumbroso S, Sultan C.

Institut National de la Sante et de la Recherche Medicale, INSERM U439, Pathologie Moleculaire des Recepteurs Nucleaires, Montpellier,France.

We previously described an androgen receptor (AR) point mutation located in the DNA-binding domain (DBD), adjacent to another AR substitution. Both were observed in two unrelated families with male breast cancer (MBC) and partial androgen insensitivity syndrome. This work was designed to determine the potential role of these two residues by in vitro study of the consequences of these two substitutions on biological functions and their structural impact at the atomic level. Mutant ARs revealed normal androgen-binding affinities and weaker DNA binding to an isolated androgen-responsive element. In cotransfection assays the mutant ARs displayed a reduced transactivation efficiency at 0.3 x 10(-10) M. Neither binding to an estrogen-responsive element nor transactivation efficiency of an ERE reporter gene was observed. Molecular modeling revealed that Arg607 and Arg608 were partially surface-exposed and located in adjacent areas in the AR-DBD complex with DNA. This is in favor of a protein-protein interaction. It is conceivable that such an interaction could be affected by mutation of one of these two arginines.

PMID: 9220020 [PubMed - indexed for MEDLINE]