a gene involved in DNA repair. In other examples, the disclosure relates to methods for treating cancer patients involving administration of an anti-PARP therapy to a patient characterized by an absence of a mutation in a gene involved in homologous recombination. In examples, the disclosure relates to methods for treating cancer patients involving administration of an anti-PARP therapy to a patient having a cancer characterized by an absence of a mutation in BRCA1 or BRCA2. In examples, the disclosure relates to methods for treating cancer patients involving administration of an anti-PARP therapy to a patient having a cancer characterized by an absence of a mutation in a gene involved in homologous recombination.

**[0005a]** In one aspect, there is provided use of niraparib in the manufacture of an oral medicament for treating platinum-sensitive ovarian cancer, fallopian tube cancer or peritoneal cancer in a patient, wherein the patient is characterized by previous treatment with a platinum-based therapy and an absence of homologous recombination deficiency (HRD).

**[0006]** In examples, the anti-PARP therapy is administered at a dose equivalent to about 100 mg, about 200 mg, or about 300 mg of niraparib or a salt or derivative thereof. In certain examples, the anti-PARP therapy is administered at a dose equivalent to about 100 mg of niraparib or a salt or derivative thereof. In certain examples, the anti-PARP therapy is administered at a dose equivalent to about 200 mg of niraparib or a salt or derivative thereof. In certain examples, the anti-PARP therapy is administered at a dose equivalent to about 300 mg of niraparib or a salt or derivative thereof.

**[0007]** In some examples, the anti-PARP therapy is administered in a regimen determined to achieve i) prolonged progression free survival as compared to control, ii) a reduced hazard ratio for disease progression or death as compared to control, iii) prolonged overall survival as compared to control, or iv) an overall response rate of at least 30%.

**[0008]** In examples, the anti-PARP therapy comprises administration of an agent that inhibits PARP-1 and/or PARP-2. In some examples, the agent is a small molecule, a nucleic acid, a polypeptide (e.g., an antibody), a carbohydrate, a lipid, a metal, or a toxin. In related examples, the agent is ABT-767, AZD 2461, BGB-290, BGP 15, CEP 9722, E7016, E7449, fluzoparib, INO1001, JPI 289, MP 124, niraparib, olaparib, ONO2231, rucaparib, SC 101914, talazoparib, veliparib, WW 46, or salts or derivatives thereof. In some related examples, the agent is niraparib, olaparib, rucaparib, talazoparib, veliparib, or salts or derivatives thereof. In certain examples, the agent is niraparib or a salt or derivative thereof. In certain examples, the agent is

## CLAIMS

1. Use of niraparib in the manufacture of an oral medicament for treating platinum-sensitive ovarian cancer, fallopian tube cancer or peritoneal cancer in a patient, wherein the patient is characterized by previous treatment with a platinum-based therapy and an absence of homologous recombination deficiency (HRD).

2. The use of claim 1, wherein the patient is characterized by an absence of a germline or somatic mutation in BRCA1 and/or BRCA2.

3. The use of claims 1 or 2, wherein the patient has a tumor with a negative HRD status.

4. The use of any one of claims 1-3, wherein the niraparib is prepared in the form of a tosylate monohydrate.

5. The use of claim 4, wherein the niraparib is to be administered as a maintenance therapy following complete or partial response to at least one platinum-based therapy.

6. The use of any one of claims 1-5, wherein the cancer is a recurrent cancer.