ANTI-PD-1 ANTIBODIES AND THEIR USES

1. CROSS REFERENCE TO RELATED APPLICATIONS

[0001] This application claims the benefit under 35 U.S.C. § 119(e) of U. S. Provisional Application no. 62/394,314, filed September 14, 2016, the contents of which are incorporated herein in its entirety by reference thereto.

2. SEQUENCE LISTING

[0002] The instant application contains a Sequence Listing which has been submitted electronically in ASCII format and is hereby incorporated by reference in its entirety. Said ASCII copy, created on August 10, 2017, is named 381493-327WO_SL.txt and is 33,195 bytes in size.

3. TECHNICAL FIELD

[0003] The present application pertains to, among other things, novel anti-PD-1 antibodies, compositions including the new antibodies, nucleic acids encoding the antibodies, and methods of making and using the same.

4. BACKGROUND

[0004] Cancer therapies comprise a wide range of therapeutic approaches, including surgery, radiation, and chemotherapy. While the various approaches allow a broad selection of treatments to be available to the medical practitioner to treat the cancer, existing therapeutics suffer from a number of disadvantages, such as a lack of selectivity of targeting cancer cells over normal, healthy cells, and the development of resistance by the cancer to the treatment.

[0005] Recent approaches based on targeted therapeutics, which interfere with cellular processes of cancer cells preferentially over normal cells, have led to chemotherapeutic regimens with fewer side effects as compared to non-targeted therapies such as radiation treatment.

[0006] Cancer immunotherapy has emerged as a promising therapeutic approach to complement existing standards of care. *See*, *e.g.*, Miller, et al. Cancer Cell, 27, 439-449 (2015). Such immunotherapy approaches include the development of antibodies used to modulate the immune system to kill cancer cells.

[0007] For example, interaction of PD-1, a type I cell surface receptor, with either of its two ligands, PD-L1 or PD-L2, results in a dominant negative checkpoint signal that limits subsequent antigen receptor-driven cellular activation. The ligands for PD-1 are differentially expressed on various