★★★ ＜第32回知的財産翻訳検定試験【第17回和文英訳】＞ ★★★
≪１級課題 -バイオテクノロジー-≫

【問 1】
[0004]
On the other hand, silkworms are classified based on their life cycle into univoltine strains, bivoltine strains, and multivoltine strains. A univoltine strain refers to a strain which develops into an adult insect once a year when bred under natural conditions. Many strains of silkworms are univoltine strains. Univoltine silkworm strains are known to diapause in the egg stage. This characteristic is retained even after the egg has been microinjected, and, therefore, to generate genetically modified silkworms in a short period of time without allowing the eggs to diapause, mutant strains that do not diapause or multivoltine strains, which are non-diapausing strains, are typically used. However, since these strains are laboratory strains, there are issues in terms of metric traits and the like, and to utilize silkworms industrially as hosts in mass production systems, it was necessary to obtain non-diapausing eggs from practical strains which are non-diapausing strains.

【問 2】
Regarding the method of the present invention for producing an extract for cell-free protein synthesis, the present method is characterized in that the middle silk gland of a silkworm strain that does not specifically produce fibroin can readily be removed, even by using a previously reported method as the method for removing the middle silk gland of a wild-type silkworm strain (see JP 2007-210902 A), compared with conventional methods for producing extracts using the posterior silk gland or fat bodies. Moreover, as mentioned below, the inventors developed a method for removing the middle silk gland of a silkworm strain that does not specifically produce fibroin in a shorter period of time compared with the conventional methods.

【問 3】
AML-MT cells were cultured in the presence of Gnetin C for 24 hours and then stained with Annexin-FITC, followed by flow cytometry analysis. In addition, the cells attached to glass slides using a cytospin were stained with Giemsa, and cell morphology was evaluated with a light microscope. AML-MT cells were cultured in the presence of Gnetin C at the indicated concentration for 12 hours, and the cells stained with MitoCapture were observed with a fluorescence microscope. Due to the ability of Gnetin C to inhibit cell proliferation in the AML cells, typical morphological signs of apoptosis including cell membrane protrusions, condensed chromatin, nucleus fragmentation, and a trace amount of condensed basophilic cytoplasm are observed in the Annexin-V staining assay and the Giemsa staining. These signs are similar to those in the apoptosis induction observed in patient-derived AML cells. Interestingly, a significant reduction in membrane potential was observed in the Gnetin C-treated cells. This suggests that the cell apoptosis induced by Gnetin C is caused by mitochondrial membrane dysfunction.

【問 4】
Claims:

1. A function-improving drug for an immune-exhausted $CD8^+$ T cell, comprising a biguanide antidiabetic drug selected from the group consisting of phenformin, buformin, and metformin.

2. The function-improving drug according to Claim 1, wherein cytokine-producing ability of an immune-exhausted $CD8^+$ T cell is recovered.

3. The function-improving drug according to Claim 2, wherein the immune-exhausted T cell expresses an exhaustion marker, and the cytokine-producing ability of an immune-exhausted $CD8^+$ T cell expressing the exhaustion marker is recovered.

4. The function-improving drug according to any one of Claims 1-4, wherein apoptosis of a $CD8^+$ T cell is suppressed.

5. An anti-tumor therapeutic agent for a tumor patient with immune-exhaustion, comprising, as an active ingredient, the function-improving drug according to any one of Claims 1-4.

6. The therapeutic agent according to Claim 5, wherein the immune-exhaustion is caused by expression of an exhaustion marker of a $CD8^+$ T cell.

7. The therapeutic agent according to Claim 6, wherein the exhaustion marker is selected from the group consisting of Programmed cell death protein 1 (PD-1) and T-cell membrane protein 3 (Tim-3).

8. The therapeutic agent according to any one of Claims 5-7, wherein the active ingredient is administered to a tumor patient not having received chemical therapy or radiation therapy for a tumor.