CYCLODEXTRIN NEWS

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HPBCD as Anticancer Drug
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A novel application field was discovered by the group of Prof. Arima (including also the Szejtli-Prize winner Prof. K. Motoyama): HPBCD was found effective in inhibition of leukemic cell proliferation at various leukemic cell lines [1]. The working hypothesis was that

- cholesterol plays an important role in the cell proliferation of various cancer cells
- agents modulating the cholesterol homeostasis might have anticancer effect
- HPBCD is well known for its cholesterol binding activity
- HPBCD might have anti-cancer effect.

HPBCD is an orphan drug approved by both FDA and EMA for the treatment of Niemann Pick type C (NPC) disease, which is a fatal cholesterol metabolism disorder. Although the exact mechanism is not known, the reduced level of cholesterol in the brain of the children suffering in this rear disease shows that HPBCD has an influence on the cholesterol accumulation and metabolism. Studying its effect against various cancers seems to be logical. The Japanese groups (Saga University, Juntendo University and Kumamoto University) started with leukemia.

The subtitles of the paper indicate the complexity of the experiments done:

- HPBCD inhibits the growth of various leukemic cell lines
- HPBCD disturbs leukemic cell cholesterol homeostasis
- Effect of HPBCD on signal transduction pathways involved in leukemia cell proliferation and survival
- HPBCD inhibits the proliferation of a tyrosine kinase inhibitor (TKI)-resistant cell line
- Administration of HPBCD prolongs survival in leukemia mouse models
- HPBCD inhibits proliferation of hypoxia-adapted leukemic cells and human primary leukemic cell colony formation

As much as 14 leukemic cell lines were involved in the *in vitro* experiments. The viability of these cell lines was inhibited by HPBCD (DS ~ 3.5) in dose- and time-dependent manner. The IC_{50} values for HPBCD fall in the range of 3.9-10.1 mM in these cell lines, while in normal hepatocytes 18.7 mM value was obtained.

Several mechanisms have been postulated and evidenced experimentally. Staining with Annexin V and 7-ADD showed that apoptosis was induced in the cells by HPBCD. It was also found to hinder cell growth by inhibiting the normal cell development. In accordance with this finding some of the G_2/M cell cycle regulators were expressed in a lower extent on the effect of HPBCD treatment.

There are patients resistant to the conventional tyrosine kinase inhibitors, such as imatinib, nilotinib and dasatinib. The T3151 mutation is responsible for that. Ba/F3 leukemic cells expressing T3151 mutant were successfully treated with HPBCD with IC_{50} similar to that of a third generation tyrosine kinase inhibitor, ponatinib.

The *in vivo* experiments showed that mice transplanted with Ba/F3 BCR-ABL leukemic cells died within 28 days when treated with vehicle but survived significantly longer when treated with 200 ml HPBCD solution of 50 mM or 150 mM for 20 consecutive days 3 days after transplantation. Similar results were obtained with NOD/SCID mice intravenously transplanted with BV173 human leukemia cells. Not any sign of toxicity of the HPBCD treatment was observed even at 150 mM concentration.

To study the effect of HPBCD on the leukemic stem-like cells, K562/HA and KCL22/HA cells were treated with HPBCD. Similar IC_{50} values were obtained as in their parenteral cells showing that HPBCD is effective not only in proliferating leukemia cells but also in dormant leukemic stem cells.

In another experiment the colony formation of mononuclear cells from acute myeloid leukemia (AML) patients was inhibited in a concentration-dependent manner.

Concerning the mechanism the affinity to cholesterol which is necessary for proliferation of leukemic cells seems to be the most important factor. Similarly to methyl BCD (MeBCD, DS ~ 11), also HPBCD enhanced the release of cholesterol from leukemic cells in dose- and time-dependent manner. Interestingly, the cholesterol content of the normal hepatocytes was not decreased at the same (10 mM) HPBCD concentration, while MeBCD decreased the cholesterol content of these cells, too. This selective effect of HPBCD on cholesterol efflux might have importance in therapy.

The expression and phosphorylation status of Akt, Erk, Stat5 and Lyn signaling proteins were studied to see if the signal transduction pathways were involved in the mechanism. In BV173 leukemic cell line the phosphorylation of Akt, Stat5 and Lyn were inhibited, while that of ERK1/2 was enhanced. In K562 leukemic cell line the p-Lyn level was reduced first on the effect of HPBCD treatment, but later on recovered. The levels of ERK1/2 increased. These results proved that the signal transduction pathways are altered by HPBCD depending on the types of cells.

These results suggest that HPBCD is a potential anticancer agent: initiating cholesterol efflux selectively in leukemic cell lines cell apoptosis and cell cycle arrest were induced and certain signaling proteins were affected. HPBCD was not toxic to mice even in large dose.

As the increased endogenous cholesterol synthesis seems to be a common property of cancer, HPBCD might be effective not only in leukemia but also in other tumors.

The anticancer effect of MeBCD has been known [2-9], but the toxicity of the methylated CDs hindered their development as drugs. The discovery of the anticancer effect of the nontoxic HPBCD in leukemia is most probably the greatest hit in the cyclodextrin research in 2015. Further studies on other cancers are expected in the near future.

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QSPR, SMILES

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Phase solubility studies, Micellar system, In vitro release assays

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Antidepressant, β-cell survival, Serum insulin level and insulin sensitivity

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Phase solubility study, Molecular modeling

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Cytotoxicity, β-Cyclodextrin, Peroxides, Drug discovery

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Maximum size of particles, Fluorescent cyclodextrins, Fish, Peritoneum, Adjuvant, Microparticles, Migration

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PEGylated protein, SPRA technology

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β-CD functionalized hydrogel, Alkynylation, Hydrophilicity, Protein resistant property of hydrogel, Thiol-yne photopolymerization

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Antifungal drug, Pathogen

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Giant unilamellar vesicles, Cholesteryl esters, Treatment for hypercholesterolemia, β-Cyclodextrins, MBCD, Cholesterol depletion

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Fibroblasts, Lysosomes

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Freeze-drying, Molecular dynamic simulation, Inclusion complex

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Fungicide, Isothermal titration calorimetry (ITC), UV–VIS, NMR, Aspergillus fumigatus

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Co-precipitation method, Minimum inhibitory concentration, Minimum bactericidal concentration, Active food packaging, Antimicrobial activity

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Benzimidazole, Self-healing properties, Multi-walled carbon nanotubes, β-cyclodextrin

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Circulation of methyl-β-cyclodextrin, Microbial degradation, Polycyclic aromatic hydrocarbon

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Phenanthrene, Pyrene, Chrysene, Benzo(a)pyrene, Methyl-β-cyclodextrin, XAD4

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Triethylene tetra amine, Enhanced surface roughness

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Interfacial adhesion, Water durability, Compatibility

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β-CD, Carbon coating, Electrochemical performance, Cycling

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Lithographic processing, Adamantyl ester, Cyclohexyl 2-propyl ether, Noria, Calixarene dimer, Cyclodextrin, Pillar[5]arene, Extreme ultraviolet

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La(III), Ce(III), Eu(III), Langmuir

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SPP bonded isopropyl cyclofructan, Desfluorinated impurities, Dehalogenation impurities, Superficially porous particles, Fluorine-containing drugs, Ultrafast separations, UHPLC

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Dopamine, Uric acid, Tryptophan, Cyclic voltammetry, Differential pulse voltammetry, β-Cyclodextrin

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α-Cyclodextrin, Excitation host, Selectivity

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Catalytic activity, Oxidation overpotential, Sensitivity, Selectivity

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Contaminant of different foods and drinks, Native and methylated β-cyclodextrins, Fluorescence enhancement, Host-guest interaction, Toxin binder

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Recognize Bisphenol A, Surface modification, Sensor

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β-CD, Antipsychotic, Experimental design

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Succinic acid, Stationary phases, HILIC method, CAD

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2-Hydroxypropyl-β-CD, 2-Hydroxypropyl-γ-CD, Dual CD system, Sulfated-α-CD, Migration order

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