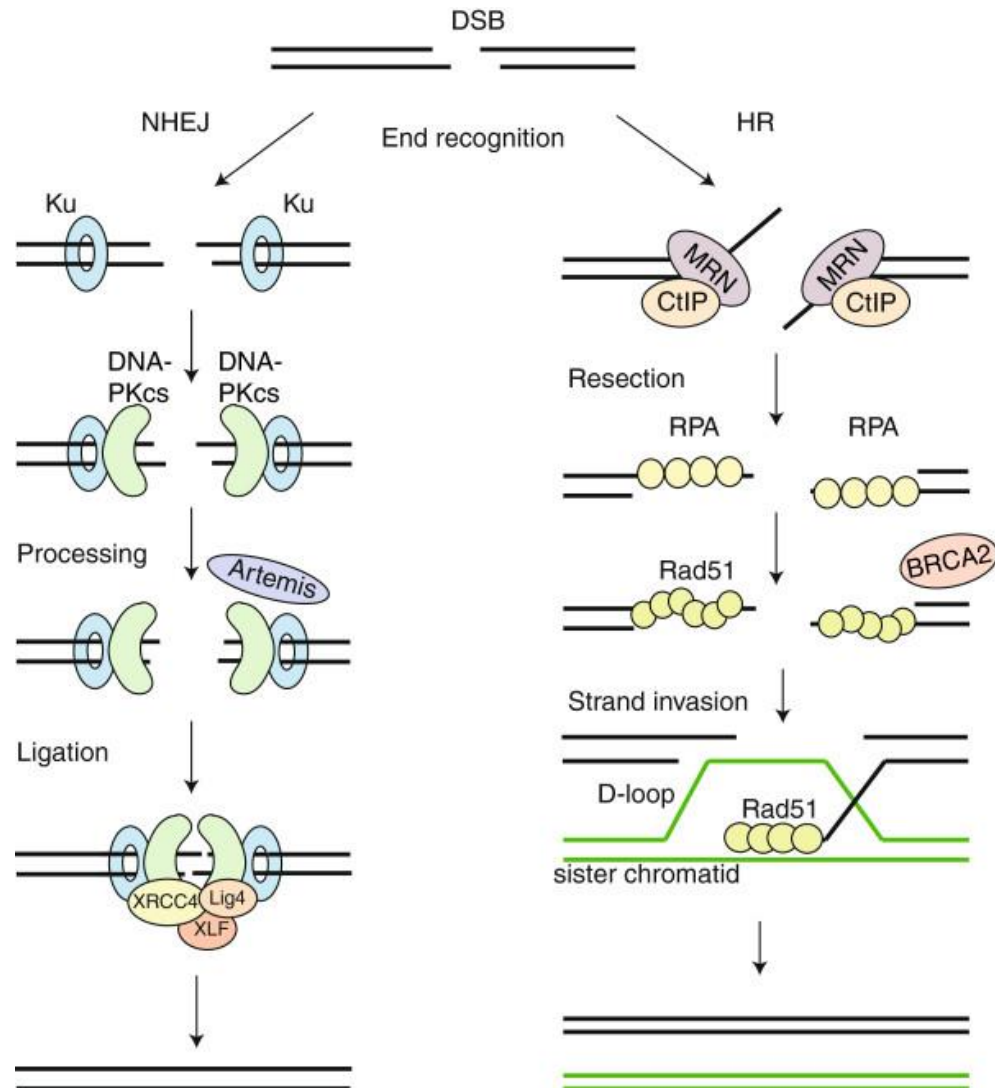


# The effect of DNA repair inhibitors on the double-strand breaks formation

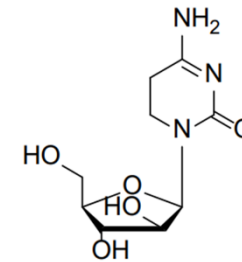
D. Shamina, A. Boreyko, V. Chausov,  
R. Kozhina, T.D. Pham, S. Tiouchik

# Main DSB repair pathways

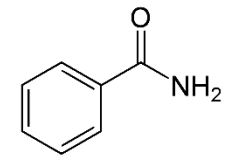


Inger B. Et al., 2012

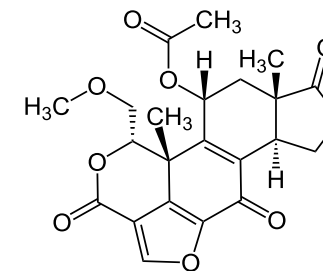
AraC



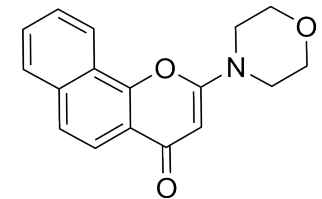
benzamide



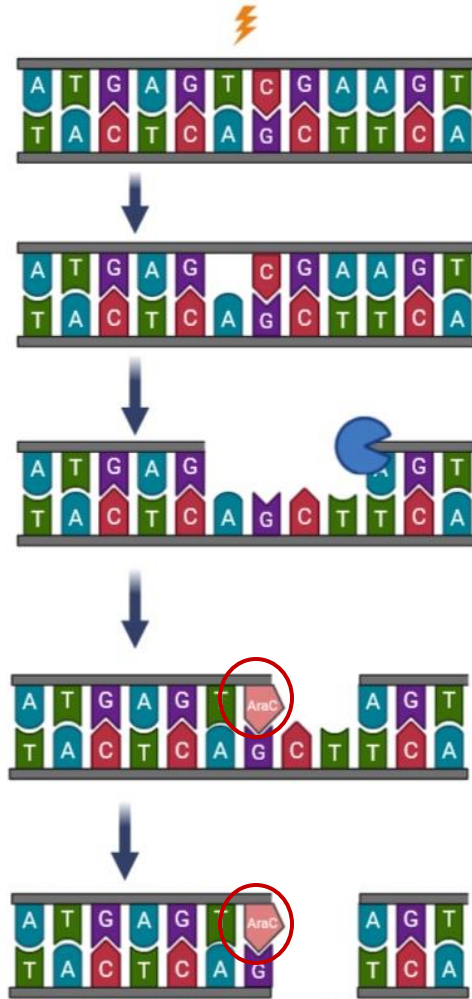
wortmannin



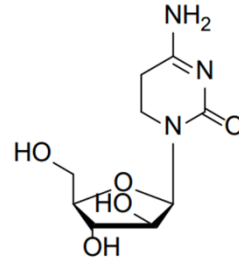
NU7026



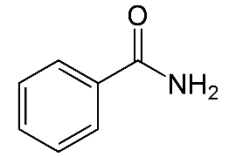
# The mechanism of AraC action



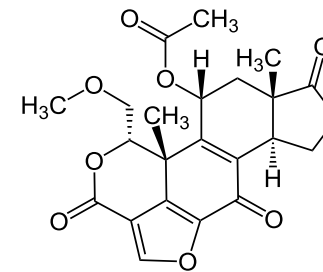
AraC



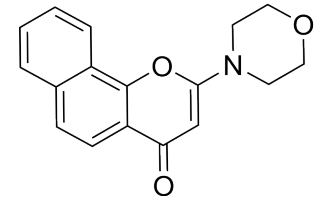
benzamide



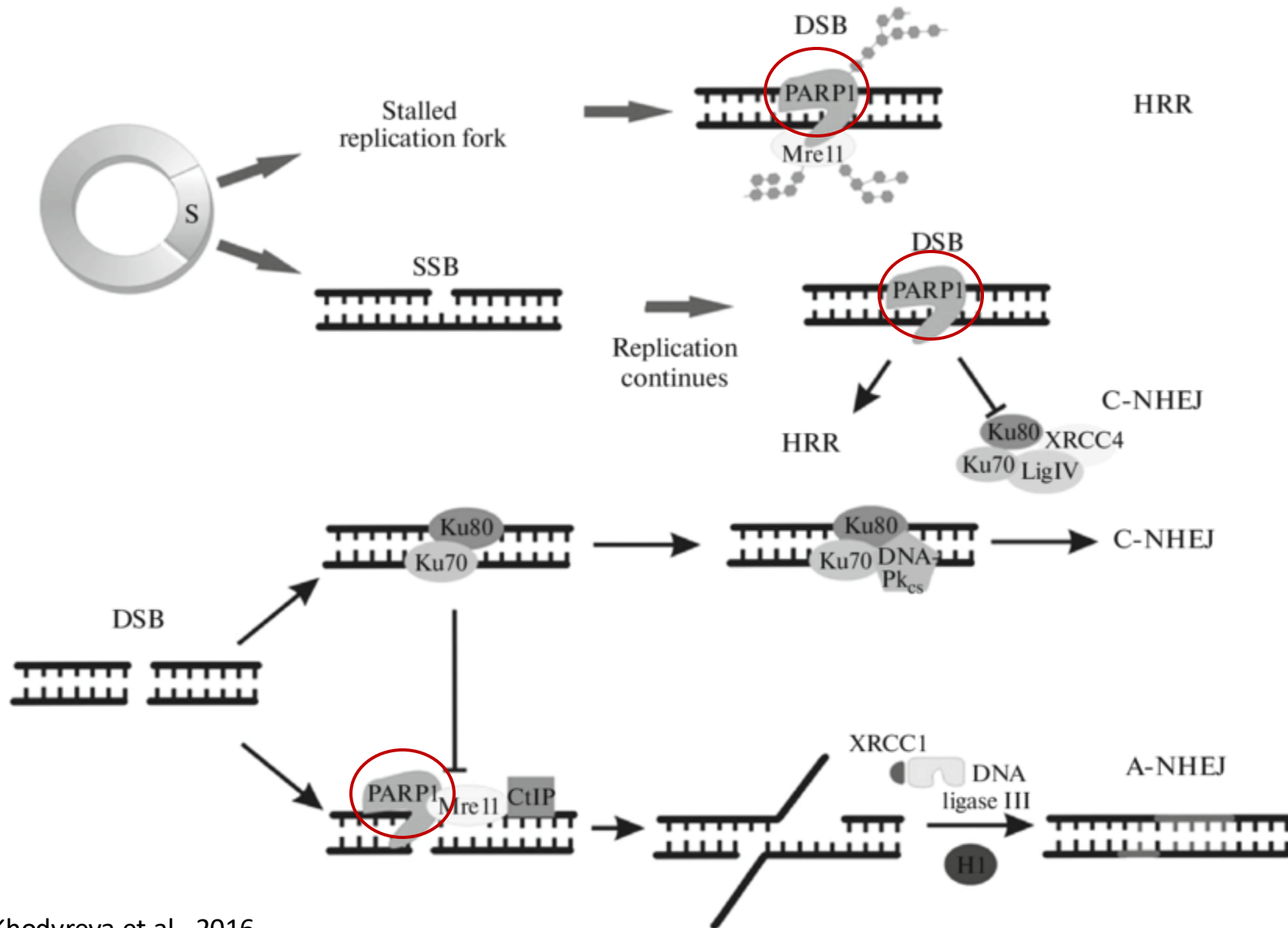
wortmannin



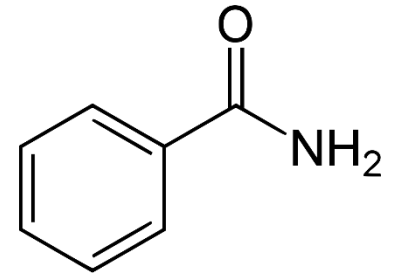
NU7026



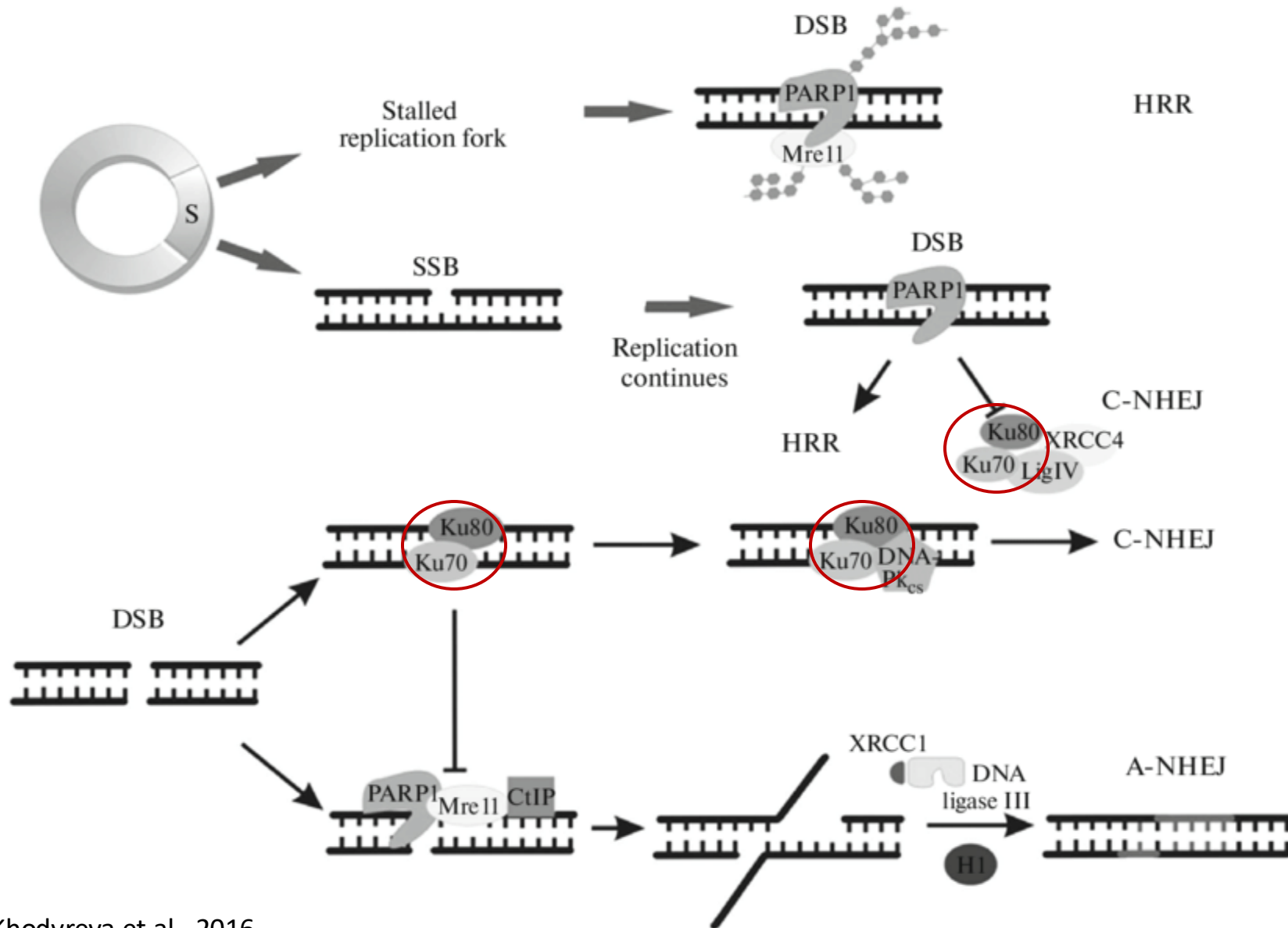
# The area of benzamide action



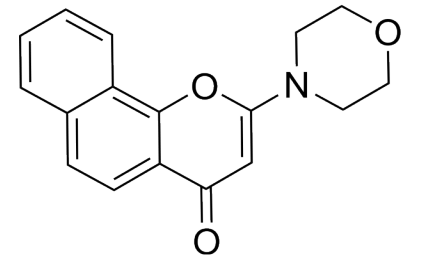
benzamide



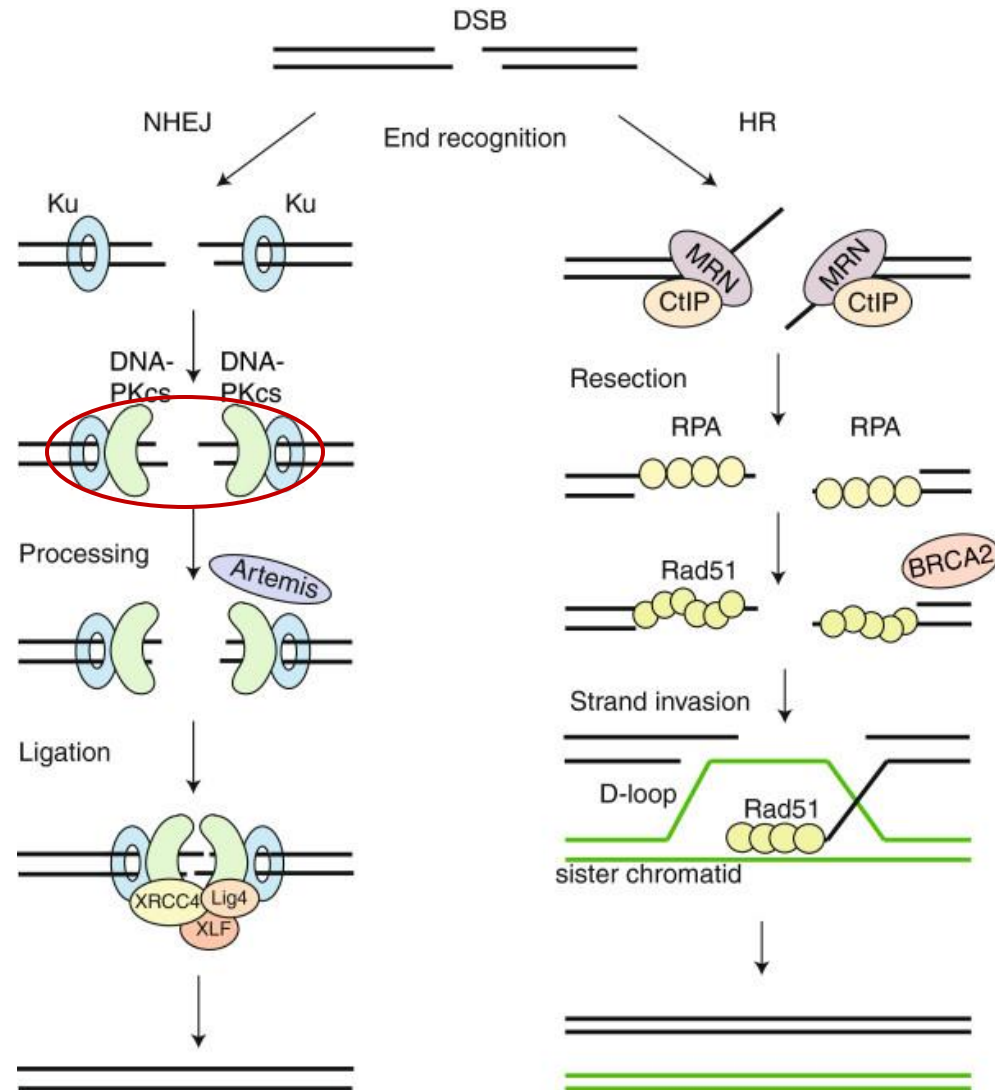
# The area of NU7026 action



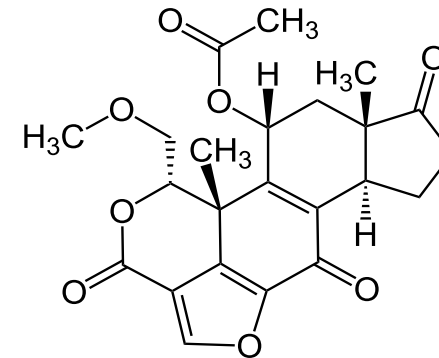
NU7026



# The area of wortmannin action



wortmannin

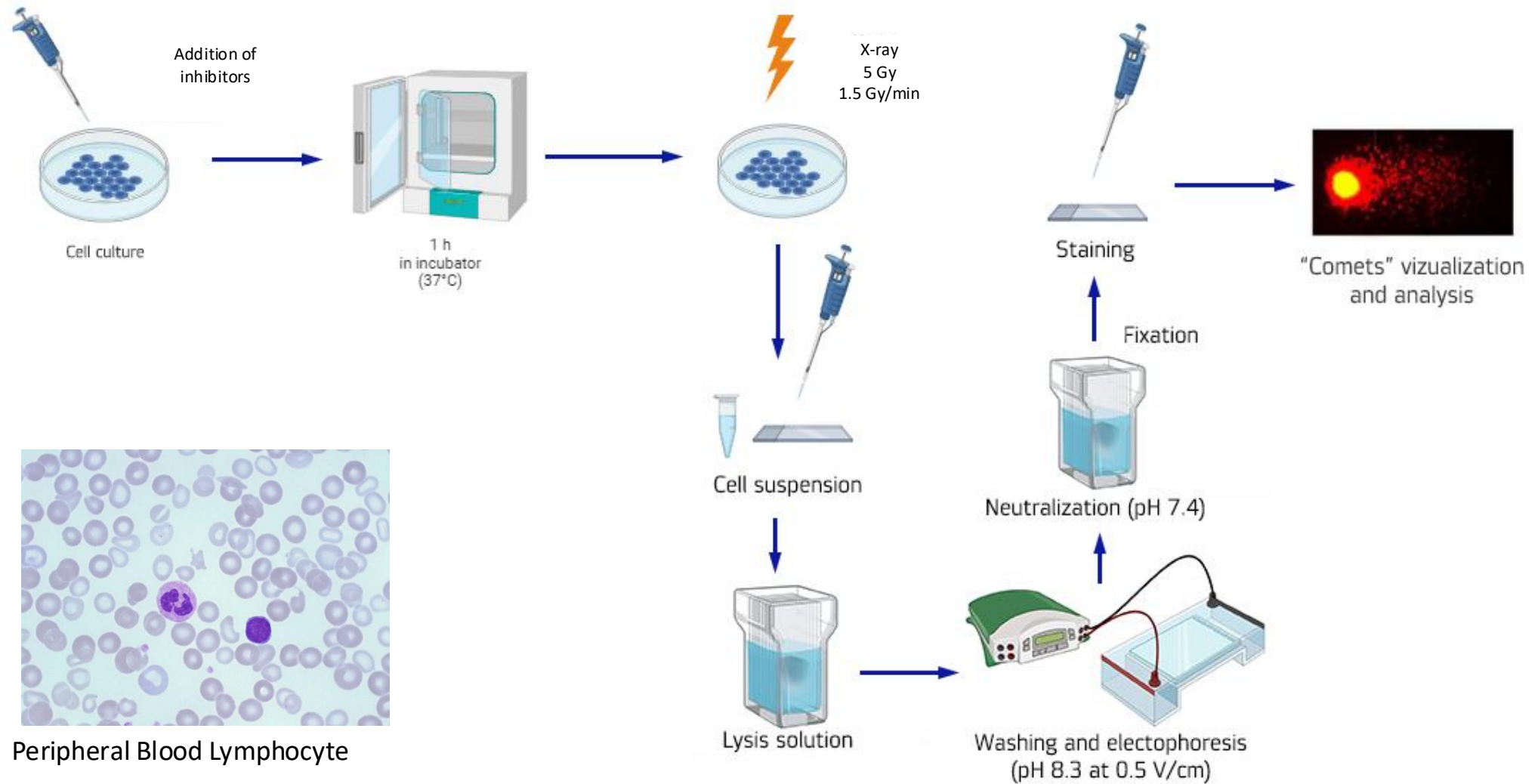


## Research goal

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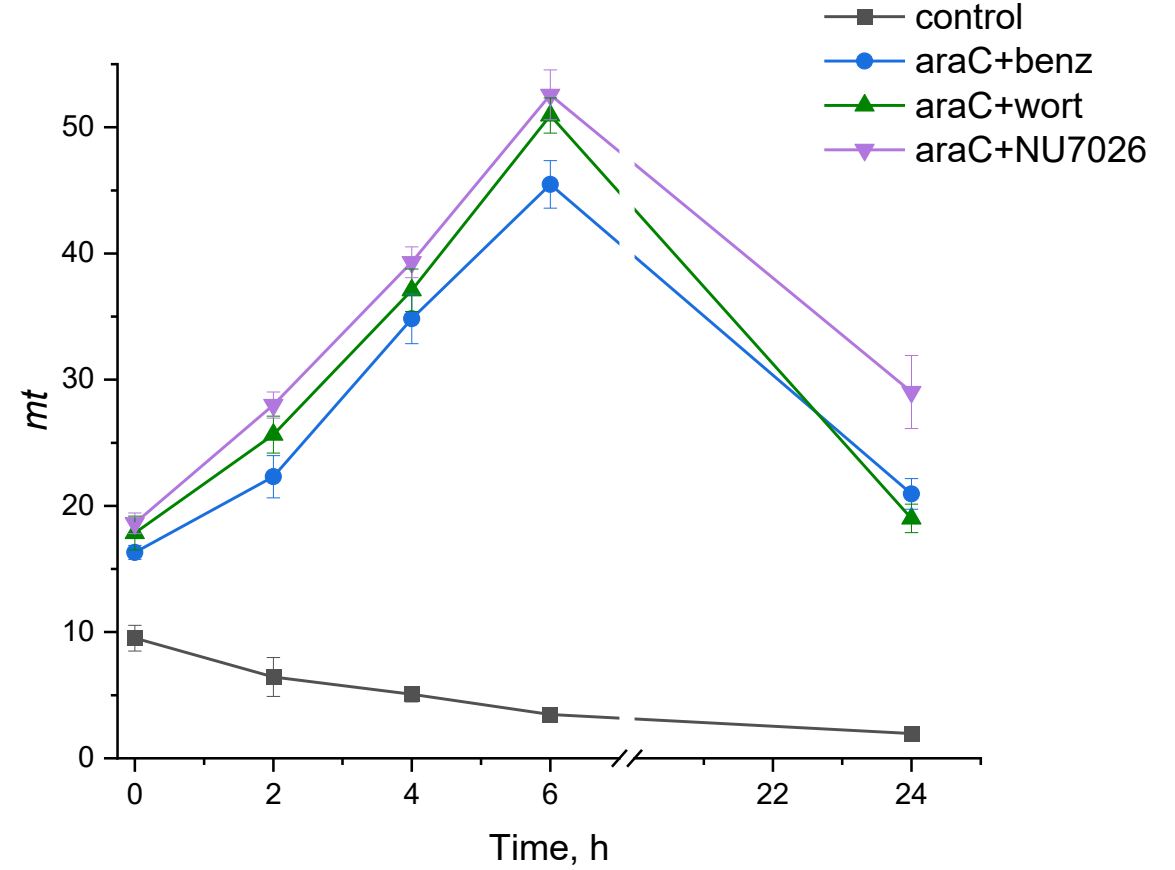
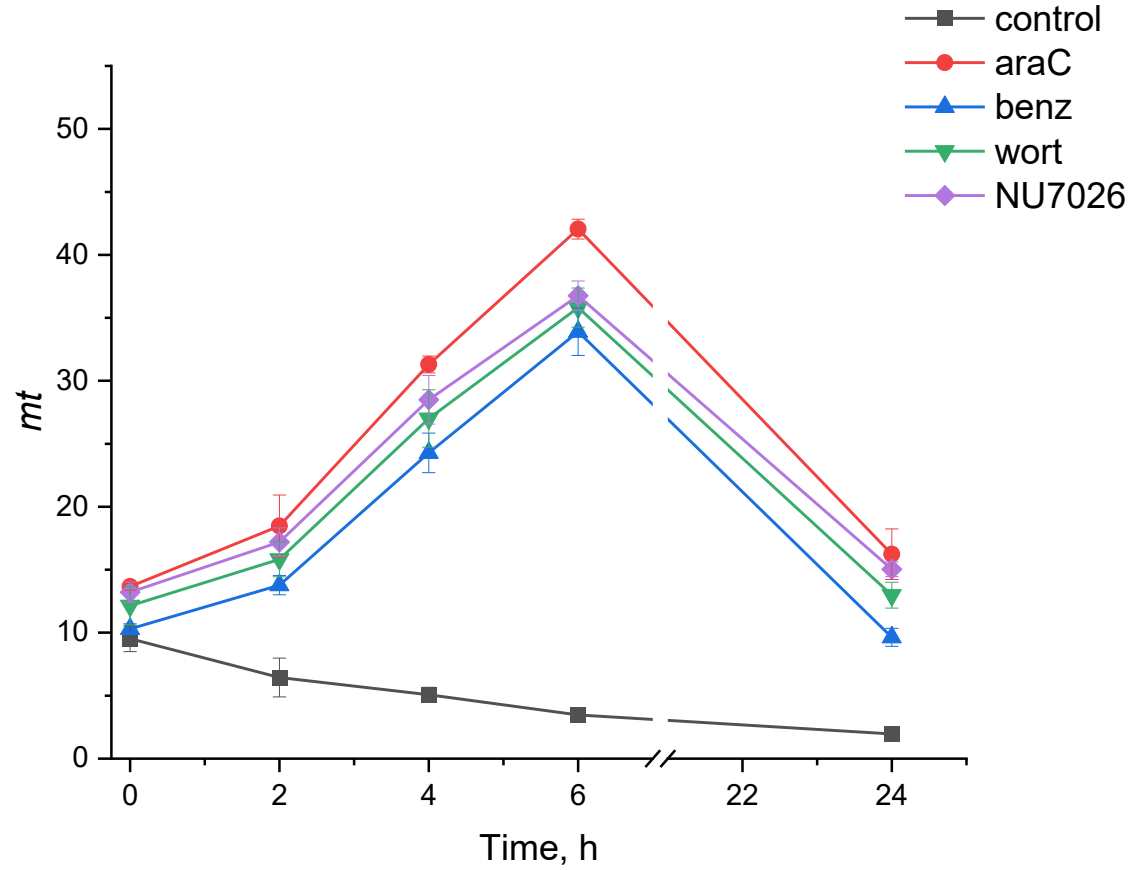
To evaluate the effect of DNA repair inhibitors AraC, benzamide, wortmannin, and NU7026 on the dynamics of radiation-induced DNA damage in human lymphocytes after X-ray irradiation.

# Materials and methods





# Results and discussion



# Summary

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- All studied DNA repair inhibitors (benzamide, wortmannin, NU7026) enhance the level of radiation-induced damage in human lymphocytes
- The combined use of these inhibitors with AraC leads to a significant synergistic effect, especially in the case of NU7026
- The data obtained confirm the promising strategy of targeted inhibition of repair pathways to increase the radiation therapy's effectiveness and, potentially, radioprotection of normal tissues



Thanks  
for your  
attention!