Summary and Conclusions

- NAR and RUT showed memory enhancing potential to prevent natural time delay-induced as well as scopolamine-induced episodic memory deficits in neurocognitive models.
- Long-term treatment with DOX resulted in episodic memory deficits but not spatial in normal healthy female rats and treatment with either NAR or RUT reversed these deficits, the most effective being RUT at a tested dose of 50 mg/kg, p.o.
- Mammary carcinoma by itself did not affect the cognitive function for either episodic or spatial learning whereas DOX severely impaired memory in mammary carcinoma bearing animals for both episodic and spatial learning functions.
- Treatment with flavonoid, RUT protected from DOX-induced chemobrain for both episodic and spatial components whereas NAR was found to be ineffective to reverse the deficits associated with DOX-chemotherapy in mammary cancer rats.
- Flavonoid, RUT did not influence the antitumor activity of DOX while being effective against DOX-induced side effects, viz., chemofog, myelosuppression and organ protection etc. and further, NAR treatment also did not influence the antitumor activity of DOX.
- RUT could be promising therapeutic intervention to prevent DOX based chemotherapy-induced behavioural and cognitive dysfunction in breast cancer survivors.
- All the future promising novel candidate molecules can be effectively screened for their protective potential against chemobrain associated cognitive complications using the above established chemobrain rat model of mammary carcinoma.
Fig. 13.1. Illustration represents possible mechanisms of test flavonoids, NAR and RUT against DOX-induced chemobrain associated cognitive dysfunction.