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A MATHEMATICAL MODEL FOR AUTOCRINE REGULATION OF GONOTROPIN- RELEASING HORMONE SECRETION IN CULTURED HYPOTHALAMIC NEURONS

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Abstract. In this paper we shall discuss about pulsatile release of GnRH is also evident in vitro during perifusion of immortalized GnRH neurons (GT1-7 cells) and cultured fetal hypothalamic cells, which continue to produce bioactive GnRH for up to 2 months. The effect of GnRH agonist and antagonist analogues on neuropeptide release are consistent with the operation of an ultrashort-loop autocrine feedback mechanism that exerts both positive and negative actions that are necessary for the integrated control of GnRH secretion from the hypothalamus. The numerical has been provided to explore the effect of various parameters on the software reliability and total cost. Finally, we decide that a computational model is based on the submission portion and we result with medical report. This paper will be very useful in the future for medical field.

1. Introduction

The term "software reliability" refers to the consistency or existence of a software system with various properties. These properties include software system trustworthiness, software expense, execution time, and software stability, among others. The probability of software errors, the extent of fault occurrence, the criticality of the fault, the corresponding module with the respective fault, and other aspects of these software systems are discussed. The pre-estimation of software reliability is needed in the software development phase in order to produce the software product. Development time is calculated based on the appropriate amount of software quality assessment of software expense. There are a variety of consistency measures that confirm the software's dependability [12]. To cope with software stability, each stage of the software life cycle takes some time. The lower the software standard, the more difficult it is to manage. Models that attempt to forecast software reliability from test results are known as software reliability growth models [13]. These models attempt to provide a connection between fault detection data (i.e. test data) and well-known mathematical functions such as logarithmic or exponential functions. The degree of similarity between the test data and the mathematical equation determines the goodness of fit of these models [3]. The likelihood that software can run without failure under a given environmental condition for a given period of time

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is known as software reliability [1] One of the most critical methods of software development is software stability evaluation. SRGMs (software stability development models) have been suggested several times since 1970. There are two kinds of programme reliability models in general: deterministic and probabilistic. The deterministic one is used to figure out how many different operators and operands are in the software. Failures and fault removals are described as probabilistic events in the probabilistic model [11].

2. Logistic Growth Curve Model

In general, programme stability improves over time, and the testing phase should be seen as a development step. That is, the improvement in reliability is attributed to the elimination of flaws. As a result, under some circumstances, The formulas used to forecast economic demographic growth may also be used to forecast software efficiency. These models basically adapt a known feature to the total number of observed faults at a given time. One of them is the logistic growth curve model, which has an S-shaped curveIts mean value and strength functions are [7].

$$m\left(s\right) = \frac{x}{1 + m * \exp\left[-ys\right]} \ , \ x \ > 0, y > 0, m > 0,$$

$$a \ \left(t\right) = \ \frac{xy \exp[-ys]}{(1 + m * \exp\left[-ys\right] \ )^2}, \ x > 0, y > 0, m > 0$$

Where x represents the predicted total number of faults to be observed, and m and y represent parameters that can be determined by fitting failure results.

3. Proposed Model

The logistic growth curve model seems to be better than the other simulations based on the model discussed. The tangential function can be used to construct the model. The positive axis must be used to draw the tangential model.. It detects that as the number of faults detected decreases, reliability rises. Zero error detection equates to unlimited reliability, while zero device reliability equates to infinite flaws. The suggested model is based on the action of software reliability and hence is applicable to software reliability.

There are an exponential number of flaws in the programme code at the start of research. The exact number of faults is unclear, although it is understood that there are a certain number of them. The defects are all of the same sort. Each flaw can be found independently of the others. Other parameters will be determined using the remaining number of faults and the remaining time. Each fault has the same chance of occurring. Each error that has arisen can be corrected immediately. The mean meaning function can be expressed as follows:

$$n\left(s\right) = \ \left(gu = \left(\frac{[1 - \exp(-s)]}{[1 + \exp(-\pi s)]}\right)\right) > 0 \ \ gu \ : 0 \tag{3.1}$$

The failure intensity can be expressed as

$$\alpha\left(s\right) = \frac{fn(s)}{f(s)} \tag{3.2}$$

The estimated amount of faults left in the programme is equal to the software's failure rate at time s, according to the software's failure severity at time s.

4. Applications

Gonadal steroids, which have beneficial and detrimental effects on the hypothalamus and pituitary gland, control the hierarchical secretion of GnRH and gonotropins (LH and FSH) [8]. In vitro experiments using hypothalamic slices, cultured hypothalamic cells, and immortalised GnRH neurons (GT1 cells) have been used to study GnRH secretion and control more recently [2, 4, 5, 9, 10, 14]. The ability of GT1 cells to release GnRH episodically in the absence of other cell types suggests that intrinsic factors such as autocrine neurosecretion control can play a role in pulsatile GnRH release.. The discovery that GT1-7 cells express GnRH receptors, whose agonist activation alters the sequence of pulsatile GnRH release by altering pulse frequency and amplitude [6], supports this hypothesis. The aim of the current research was to see whether autocrine modulation mediated by endogenous GnRH receptors is also present in normal GnRH neurons. The expression of GnRH receptors, as well as the effects of GnRH agonist and antagonist analogues on the dynamics of GnRH release from negative GnRH neurons, were studied using cultured foetal hypothalamic cells.

FIGURE 1. The effects of GnRH agonist and antagonist analogues on GnRH release in hypothalamic cells and GT1-7 cells in static cultures

Treatment with GnRH agonist ([D-Ala^6]Ag) and antagonist (SB-75) analogues that did not cross-react in the GnRH RIA was used to assess the role of GnRH receptor activation in neuropeptide secretion. The combined release of GnRH was determined in hypothalamic cells (fig. A) and GT1-7 cells (fig. B) during static culture and treatment with low concentrations of the two analogues. No immuno reactive GnRH was detectable in the medium of controls and treated cells at the zero time stage.

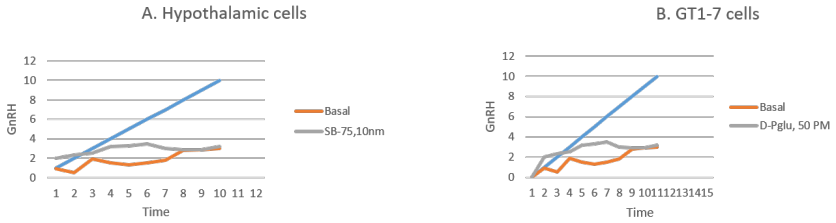
FIGURE 2. GnRH release from cultured hypothalamic neurons is pulsatile. A. B, secretory profile of GnRH extracted from every fifth sample in the data set seen in (A). C, GnRH secretory profile calculated as total development over 5 minutes. D, GnRH biological activity released by pituitary cells moving through perifusion media.

When perfused in the absence of exogenous factors, cultured foetal hypothalamic cells continuously released pulsatile GnRH (A). The GnRH secretory profile obtained by plotting every fifth measurement from the original data set was similar to the GnRH secretory profile generated by all data points. (B.) The curve for GnRH release obtained by measuring the cumulative value during 5-min collections and the curve extracted from the original data set were both quite similar (C). By steering the perifusion medium from hypothalamic cells into a downstream champer containing anterior pituitary cells, the biological activity of GnRH produced by hypothalamic cells was assessed. Basel LH release was poor before the interaction with a champer containing hypothalamic cells, but it greatly increased after the connection was made.

FIGURE 3. The effects of agonist treatment on GnRH release from perifused hypothalamic cells are dose dependent.

GnRH agonist and antagonist analogues have more complex effects on the secretory pattern when studied in perifused hypothalamic cultures and GT1-7 cells. The pulse rate was further reduced when perifused hypothalamic cells were exposed to 100nm[D-Ala6Ag]. In the GnRH RIA, the GnRH agonist analogue did not cross-react over a large spectrum of concentrations, as seen in figure c.

FIGURE 4. GnRH receptor inhibition inhibits the release of GnRH from perifused hypothalamic cells. A, The GnRH receptor antagonist, SB-75(10nm), was used for the following 3-hour recording cycle after 2 hours of basal GnRH release (open circles) (closed circle).



In static societies, the above answer was consistent with the above observation. The average GnRH level in perifused hypothalamic cells. During therapy with the potent GnRH antagonist, GnRH release was observed in six of these studies.. Treatment with the Antag increased GnRH release in perifused GT1-7 neurons, which was accompanied by a temporary peak response (B). In the GnRH RIA, none of the GnRH antagonists cross-reacted over a broad spectrum of concentrations, as seen in figure C.

5. Mathematical Result

FIGURE 5.

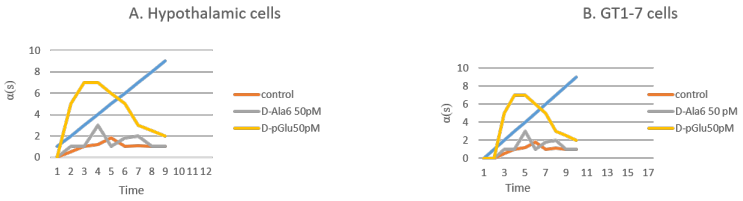


FIGURE 6.

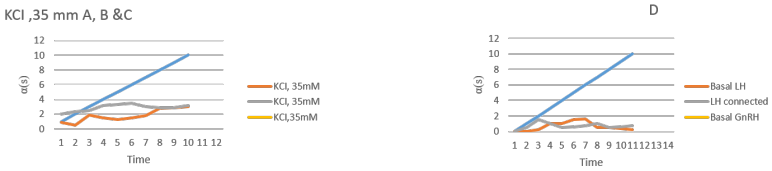


FIGURE 7.

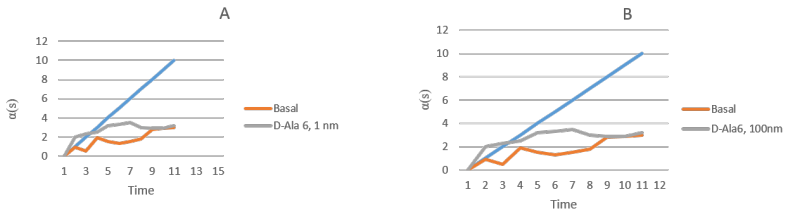
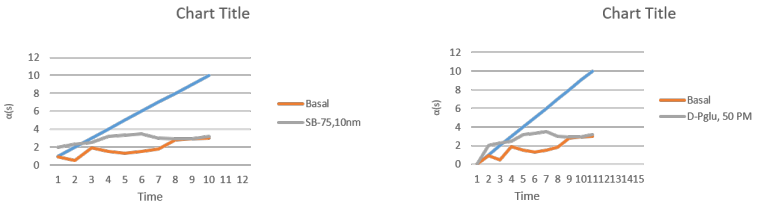


FIGURE 8.



6. Conclusion

The present findings, and observations in other experimental models, indicate that normal GnRH producing neurons coopers GnRH and GnRH recepters and exhibit spontaneous electrical activity that controls Basel GnRH release. These characteristics are appropriate for the operation of an oscillator that is controlled by both positive and negative ultrashort-loop autocrine feedback mechanisms. Finally, we conclude that a mathematical model is coinciding with application part and conclusion is compared with model report. This paper will be very useful for medical field in future.

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Conflict of Interests. The authors declare that there is no conflict of interests.

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