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Method for Analyzing the Multi Modes Acetylcholine Receptor Channel

---- H2 control strategy ----

H, Hirayama., N, Kitagawa., Y, Okita and T, Kazui.

Department of Public Health Asahikawa medical college
The Graduate School of Shizuoka university
The First Department of Surgery Hamamatsu medical college.

We proposed a method for analyzing the performance of multi modal Acetylcholine receptor complexes in the synaptic transmission of the central nervous system. The acetyl choline-receptor binding and activating processes of these complexes consisted of one Ach -receptor complex, two Achs -receptor complex and Ach free receptor. Each of them have activated form or inactivated form. Ach was set as control input. For the H2 control, we induced the differential equations for the state variables and estimator of the system which were expressed by Riccati equations. By solving the differential equations linked by the Riccati equation, the transient changes in the concentration of the Ach-receptor complexes were computed numerically. The present investigation will be available for Ach -receptor channel function of the central nervous system.

Central nervous system, Acetyl-choline, Receptor, Channel H2 control, Riccati equation

神経伝達物質アセチルコリンチャンネルの制御特性解析

-----H2 制御 ------

平山博史, 北川敬之, *沖田善光, **数井暉久

旭川市西神楽４-５旭川医科大学 公衆衛生学講座
（電話0166-65-2111、内2411） E mail hirayama@asahikawa-med.ac.jp
* 静岡大学大学院電子科学研究施設
**浜松医科大学外科学第一講座

中枢神経系のシナプス間隙伝達物質の１つであるアセチルコリンAch チャンネルの動的特性と制御機能を解析した。Ach受容体チャンネルの開閉を６つの状態モデルで表現した。すなわちAch 分子が１こ、または２こ結合してチャンネルが開いている状態と閉じている状態、および Achが結合していない状態での開閉を６個の状態方程式で記述した。制御入力としてはAchを設定した。これに対してまず線形システム解析をし、つぎにH２制御をおこなった。制御と観測の２つの方程式群はリカッチ方程式で連結した。質量保存則を用いて合計１０個の微分方程式を数値計算で解いた。 Ach 分子が１こ、または２こ結合した受容体分子の過渡的変動を計算することが可能であった。 本研究は中枢神経系伝達物質の受容体結合過程の制御特性を評価すううえで有用である。

中枢神経系. アセチルコリン. チャンネル. 受容体. Ｈ２ 制御. リッカチ方程式

1. Introduction

Acetylcholine is a kind of biochemical information transmitter released at the pre synaptic bottom on the end plate of muscle (Fig 1-a). Binding of Ach molecule on the receptor on the target cellular membrane evokes conformational change in the channel molecules (Fig 1-b. The electron microscopic illustration of Ach binding channel. It is consisted of four to five subunit which molecular conformations are similar but a little difference Fig 1-c). The precise structure of the molecular conformation has been disclosed although we have scarcely find controlling strategy how the Ach evoked channels should be organized.

In the present investigation, we propose H2 control for temporal feature of multiple states Ach channel. The modeling basis on the patch clamp experiments reported by Colquhoun and Sakmann (1985).

Fig 1-a

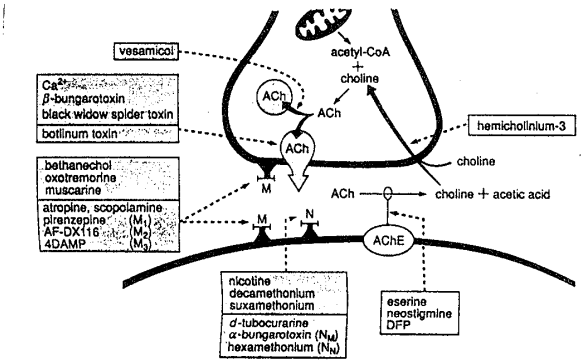


Fig 1-b

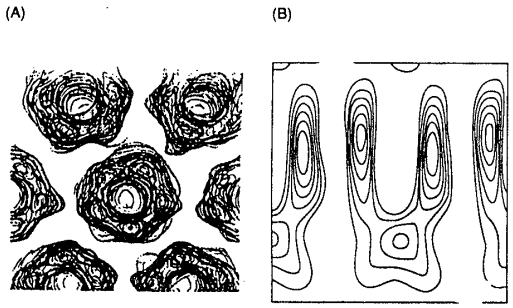
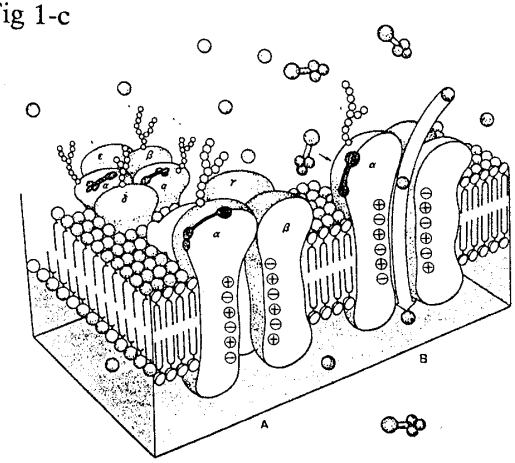


Fig 1-c



2. Reported experimental results.

Colquhoun D and Sakmann B (1985) have investigated single channel currents activated by acetyl choline and and its analogues at the frog muscle end-plate. They used the patch clamping method to analyze only one isolated behavior (namely the temporal and sequential change in the electrical current evoked by channel gating through binding acetyl choline molecule to the channel receptors on the membrane of the end plate). They report

1. Individual activations of the channel (opening the channel and leading to the current in flow) which are named bursts were interrupted by short closed periods.

2. The temporal distribution of such duration was composed of a major fast component (named short gaps) and a minor slower component (named intermediate gap).

3. The mean duration of both short and intermediate gaps depended on the nature of the agonist. For the short gaps, the mean duration (μs) were 20μs for Ach (acetyl choline). The mean number of short gaps per burst was 1.9 for Ach.

4. The mean number of short gaps per burst and the mean number per unit open time depended on the nature of the agonist. These parameters little depended on agonist concentration or the membrane potential for Ach.

5. Partially open channels (sub conductance state) were clearly resolved rarely (0.4 % of gaps within bursts) but regularly. Conductance of 18% (most commonly) and 71 % of the main value were found. The most short gaps were full closures.

6. The distribution of burst length had two components. The faster component represented mainly isolated short openings. Such short openings were frequently observed at low concentration of agonist Ach. The slower component represented bursts of longer openings. More than 85 % of activations were of this type. This corresponds to the channel life time found by noise analysis.

7. The frequency of channel openings increased slightly with hyperpolarization.

8. The short gaps during activations were not affected when the concentration H ion and Ca ion out side of the end plate membrane were reduced to 10% of the normal.

9. Reduction of Ca ion out side of the end plate membrane to 10% of normal elevated the single channel conductance by 50%. Such changes significantly augmented the number of intermediate gaps.

10. temporal asymmetry was not observed in the bursts of openings. The length of successive apparent open times at low concentration of Suberyldi choline (SubCh) had positive correlations. However, there was no correlation between the bursts lengths.

11. The component of brief openings at low concentrations may originates from openings of singly occupied channels.

12. Short gaps within bursts act as if the channel is composed of an allosteric molecular unit. They behave like multiple openings of the doubly occupied channel before dissociation occurs.

3. Consideration for modeling the Ach channel.

Colquhoun and Sakman (1985) inferred the multiple states of the Ach channel gating. They supposed that the number of shut states must be at least the number of exponential components that were needed to fit distribution of all shut times (Colquhoun and Hawkes 1981, 1982) under the assumption that

1. The channel exist in several more discrete states
2. The channel is memory less (Markov assumptions that underline law of mass action calculation).
3. the rate constants for transitions between states are constant and do not vary with time.

Based on their experimental observation, they speculated that there are at least three kinds of shut state. Moreover there are desensitized states a and blocked states.

For high agonist concentrations, there may be two more exponentials for accounting the putative desensitized states. For blocked channel state, a sixth exponential may be required.

By the same consideration, the number of opening states can be predicted from the number of components fitted to the distribution of open times. These were measured only as apparent opening and the distribution of total open times per burst. From their observation, there must be at least two kinds of opening states. Consequently they speculated that three shut and two open states.

3-1. Nature of the shut states.

Colquhoun and Sakmann (1985) have taken into consideration of their experimental data

1. the mean duration of the gaps within burst depended on the nature of agonist
2. neither the mean duration nor the frequency of such gaps depended on either agonist concentration or on the membrane potential.

3-1-1. Normal shut channels.

Lots of biochemical reports suggested that there are two binding sites for agonists per ion channel. (Karlin A., Cox R., Kaldany R-R. Lobel P and Holtzman E. 1983. Cold spring Harbor Symposium on Quantitative Biology. vol 48. pp 1-8. 1983. The arrangement of functions of the channels of the acetylcholine receptor of Torpedo electric tissue.) This is consistent to the electro physiological evidence concerning the cooperativity of the responses. Hence, they speculated that there must be at least three shut states of the normal channel in which the channel binds to zero, one and two agonist molecules. The gaps within bursts could be caused by oscillation between the open states and the shut states.

3-1-2. Ion channel blockages by the agonist

The most reliable possibility is that brief gaps is evoked by transient blockages of the open ion channel by the agonist itself. The lack of agonist-concentration dependency in the frequency of short and intermediate shut periods rules out the possibility that these shut periods result from block by the agonist molecules themselves.

3-2. The nature of the brief opening component.

The brief component in the distribution of apparent open times or of bursts lengths could never be suspected from the noise analysis.

3-2-1. Two kinds of channel.

Colquhoun and Sakmann (1985), discussed that there could be two independent types of channel, one is producing mainly single brief openings, the other is producing burst of longer openings. This can not be ruled out. it is quite compatible with the observed correlation between open times and the observed concentration dependence of the relative number of short openings with Sub Ch could result from the two channels having different concentration-response curves or different desensitization characteristics. On the other hand, the relative number of short openings reasonably could be reproduced from patch to patch. To observing such evidences, an even distribution of the two kinds of channel on the membrane and a large number of channel in each patch are required. The amplitude of the brief openings apparently seemed to be similar to that of the long openings. Both have the amplitude expected for junctional type channels.

3-2-2. Singly occupied channel openings.

Karlin 91967) verified that channel might open with only one agonist molecule that has been bound to the channel. This must be the origin of the short openings. Hence, the ratio of the areas for the slow and fast components of the burst length distribution should increase approximately linearly with agonist concentration. even if there were two non equivalent subunits. Colquhoun and Sakmann observed a linear relationship at low concentration of SubCh. At high concentration, however, the relative number of short openings does not fall as quickly as predicted. They inferred that it could be that the rather small brief component which is less than 15% that remain at higher concentration arises in some other way.

3-2-3. Sub conductance states.

The sub conductance states have been observed in only a very low frequency. There were two kinds of sub conductance level characterized by two discrete values. The relative magnitude of the sub conductance level were with mean amplitude of 18% and 71% of the main conductance state. The less frequently observed 71% amplitude state is comparable in its conductance to those reported for the non-synaptic channel in frog muscle. (Neher and Sakmann 1976. in J. Physiology. vol 258. pp 705-729. Nois analysis of drug induced voltage clamp currents in denervated frog muscle fibers). It may be that the channel is conformed by five helixes, one from each subunit of the receptor-channel protein. Hence, the sub conductance level may be related to the barrel-like holes with several different open states.

3-3. Possible mechanism.

On the basis of patch clamp experimental data, Colquhoun and Sakmann (1985) proposed a possible

mechanism of multiple states Ach channel gating current which must be founded on the Monod Jacob Wyman's allosteric modeling of the enzyme. The mechanism represented in schema is extended from Karlin (1967) (J. theoretical biology. vol 16. pp 306-320. 1967 : On the application of a plausible model of allosteric proteins to the receptor for acetycholine). This allosteric modeling is likely to account for the observed co-operativity of the channel. In the schema, the definition of the variables

R : the shut receptor-channel molecules.

R* : the open conformation

A : agonist

The transitions between the states are characterized by rate constants. In this investigation, we assumed that unoccupied channels can open. In this mechanism, oscillations between A2R a and A2R* could cause short gaps within bursts (longer gaps result if AR were reached) and AR* might represent brief openings.

Colquhoun and Sakmann inferred from their brief gaps that the mean length of brief gaps within bursts single sojourns in A2R is

$\tau f = 1 / (\beta + 2\ k\text{-}2)$

and the mean number of such gaps per long bursts will be

$nb = \beta / (2\ k\text{-}2)$

Hence, the values for β and k-2 can be estimated for each agonist. These calculations are applicable for the transitional situations of

AR <-----> A2R <---> A2R*

When the two binding sites were non-equivalent, the values of k-2 given expresses the mean of the dissociation rates from the two kinds of site.

Neither of τf and nb depended on the membrane potential. As a result, the estimates of β and k-2 also is little voltage dependence. The fact that the equilibrium constant for binding of competitive antagonists which probably bind at the same site as the agonist is not voltage dependent implies that the binding of agonist and hence k-2 is not voltage dependent.

Altought the voltage dependence of equilibrium response at low agonist concentration is similar to the voltage dependence of the mean channel life time, such result might describe the voltage dependence of binding rather than that of the opening reaction. Consequently, Colquhoun and Sakmann (1985) concluded that both of β and k-2 little depended on the voltage.

On the basis of their experimental data they calculated that β = 30600 /sec and k-2 = 8150 /sec,

the mean length of a single opening at -130 mV of the membrane potential was $1 / \alpha$ = 1.4 msec which is approximately 1/3 of the burst length. The conformational equilibrium constant was β / α = 43 and the equilibrium constant for binding was 80 μM.

These values consisted to that binding is much faster than channel opening.

4. Illustration of Ach dynamic properties from the investigation of Colquhoun and Sakmann (1985).

The following figures describes important functional properties of the Ach channel that have been disclosed by patch clamp method.

Fig 2

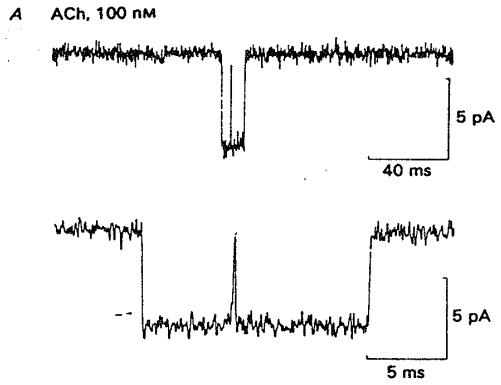


Fig 2 is an example of single channel activated elementary current by 100 nM-Ach. Upper low time resolution and lower high time resolution. This elementary current represents two resolved channel openings separated by a short closure.

Fig 3

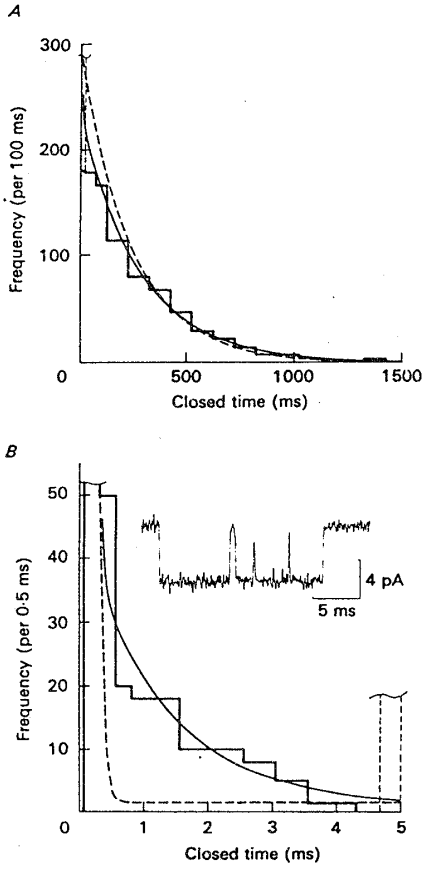


Fig 3. Distribution of the duration of shut times with 100nM-SuCh. A shows the whole distribution up to 1500 ms. B shows the intermediate component. The inset shows a channel opening consisted of three short gaps.

Fig 4

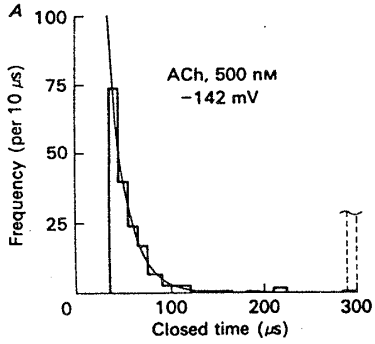


Fig 4 shows the distribution of shut times for Ach up to 300 μs to illustrate the distribution of the shortest gaps. The distribution has fitted with at least three exponential.

Fig 5

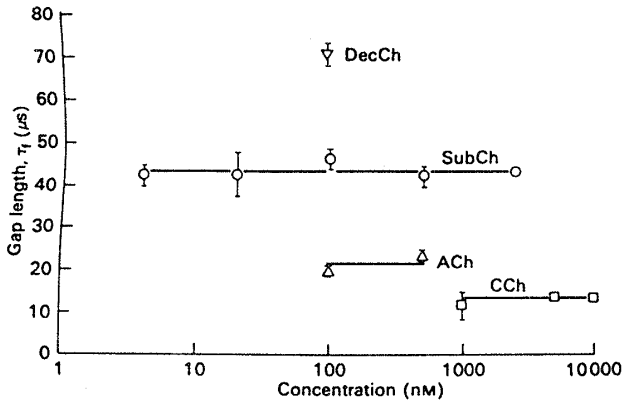
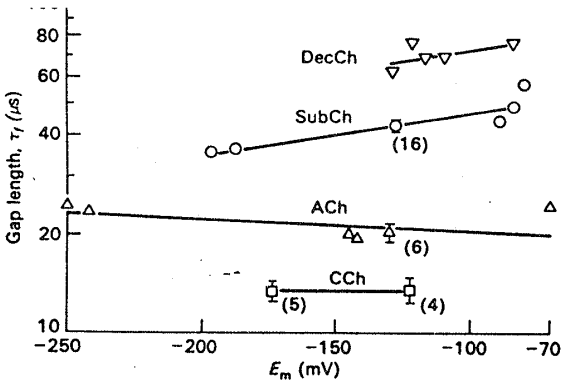


Fig 5 shows the dependence of the time constant for the brief gap component on the agonist concentration.

Fig 6 shows the time constant for the brief gap component on the membrane potential.



5. Modeling of multiple states Ach channel.

¥The system equations are

$R' = \alpha o [R^*] + k\text{-}1 [AR] - (\beta o + 2\, k1 [Ach]) R$

$AR' = 2\, k1 [Ach] [R] + \alpha 1 [AR] + 2\, k\text{-}2 [A2C] - (k\text{-}1 + \beta 1 + k2 [Ach]) [AR]$

$A2R' = k2 [Ach] [AR] + \alpha 2 [A2R^*] - (2\, k\text{-}2 + \beta 2)[A2R]$

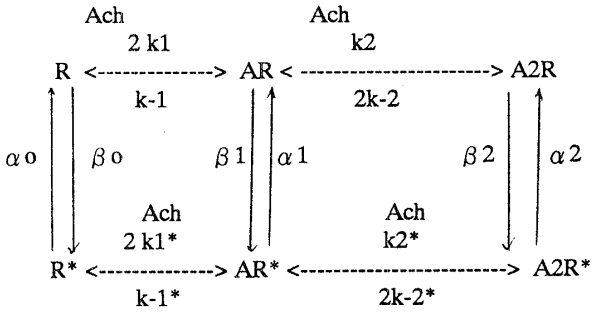
$A2R^* = \beta 2 [A2R] + k28 [Ach] [AR^*] - (\alpha 2 + 2\, k\text{-}2^*)[A2R^*]$

$AR^{*\prime} = 2\, k\text{-}2^* [A2R^*] + \beta 1 [AR] + 2\, k1^* [Ach] [R^*] - (k2^* [Ach] + \alpha 1 + k\text{-}1^*) [AR^*]$

$R^{*\prime} = \beta o [R] + k\text{-}1^* [AR^*] - (\alpha o + 2k1^* [Ach]) [R^*]$

where ' denotes differentiation with respect to time. AR, A2R* denote the amount of Ach- channel activated states per unit membrane area. kn is rate constant.

Fig 7. The possible mechanism of multiple states Ach channel.



The state equations are

X1' = a11 X1+ a12 X2 + a13 X3 +a14 X4+a15 X5+ b1 U1

X2' = a21 X1+ a22 X2 + a23 X3 +a24 X4+a25 X5 + b2 U1 + b3 U2

X3' = a31 X1+ a32 X2 + a33 X3 +a34 X4+35 X5+ b4 U2

X4' = a41 X1+ a42 X2 + a43 X3 +a44 X4+45 X5+ b5 U3

X5' = a51 X1+ a52 X2 + a53 X3 +a54 X4+a55 X5 + b6 U3 + b7 U4

where X1 = [R*], X2 =AR*, X3 = A2R*
X4 = A2R, X5 = AR due to the law of conservation. The control inputs were set as

U1 = - 2 k1* [Ach], U2 = k2* [Ach], U3 = k2 [Ach]
U4 = -2 k1 [Ach]

an are coefficient characterizing the system and bn is those for the control inputs.

For the state equation including the disturbance noise w is

$X' = A X + B1\, w + B2\, U$

where

$$B2 = \begin{matrix} b1 & 0 & 0 & 0 \\ b2 & b3 & 0 & 0 \\ 0 & b4 & 0 & 0 \\ 0 & 0 & b5 & 0 \\ 0 & 0 & b6 & b7 \end{matrix}$$

The out put vector is

$Y = C2\, X + D21\, w$

The setting of estimating vector z is

$Z = C1\, X + D12\, U$

which actual form is

z1	0	0	0	0	0		q6	0	0	0	
z2	0	0	0	0	0		0	q7	0	0	
z3	0	q2	0	0	0	x1	0	0	0	0	
z4	q1	0	0	0	0	x2	0	0	0	0	u1
z5	0	0	q3	0	0	x3	0	0	0	0	u2
z6	0	0	0	q4	0	x4	0	0	0	0	u3
z7	0	0	0	0	q5	x5	0	0	0	0	u4
z8	0	0	0	0	0		0	0	q8	0	
z9	0	0	0	0	0		0	0	0	q9	

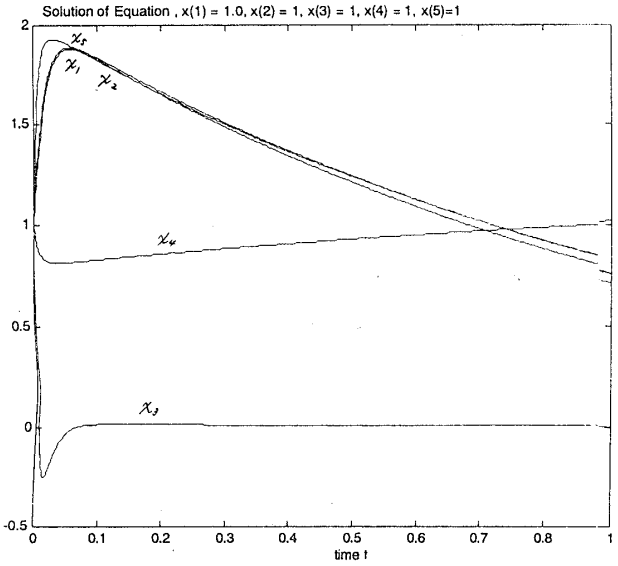
The optimal control input $U^\wedge$ is given by

$$U^\wedge = -B2^T X x^\wedge$$

and $x^\wedge$ is the state variable for the observer that satisfies

$$x^{\wedge\prime} = A x^\wedge + B2\, u^\wedge + Y C2^T (y - C2\, x^\wedge)$$

Fig 8



7. Results and Discussion.

Fig 8 shows the temporal changes in the amount of channel states X1 to X5 per unit membrane are under the control strategy of H2 control. The present method is still premature in precise determination of rate constants and coefficients characterizing the noises w. Such insufficiencies will be conquested in future our work.

9. Conclusion

Modeling Acetyl choline receptor system by multiple states channel and application of H2 control strategy will be available for evaluating the Ach induced channel kinetics.

10. References

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APPENDIX 2.

1. Standard LQR problem.

We define a dynamical system by

$$x' = A x + b2\, u \quad \text{-----(13-6)}$$

$$z = C1\, x + D12\, u \quad \text{------(13-7)}$$

We suppose that the system parameter matrices satisfy the following assumptions

A1. (A, B2) is stabilizable

A2. d12 has full column rank with [D12 $D^\perp$] unitary

A3. (C1, A) is detectable.

A4.

$$\begin{bmatrix} A - j\omega I & B2 \\ C1 & D12 \end{bmatrix}$$

has full column rank for all ω.

Find an optimal control law

$u \in L2[0,\infty)$ such that the performance criterion $\| z \|_2^2$ is minimized.

Remark 13.1 Individual assumptions.

A1 is necessary for the existence of a stabilizing control function u.

A2 states that $R = D12^* D12 = I$.

A3 enforces that the unconditional optimization problem will result in a stabilizing control law. Assumption A3 together with A1 guarantees that the input/ output stability implies the internal stability

$u \in L2$ and $z \in L2$ imply $x \in L2$ which will be shown in Lemma 13.1.

A4 is equivalent to the condition that ($D^*{}_\perp C1$, A - B2D12*C1) has no observable modes on the imaginary axis and is weaker than the popular assumption of detectability of ($D^*{}_\perp C1$, A - B2D12*C1). A4 together with the stability of (A,B2) guarantees by **Corollary 12.7** that the following Hamiltonian matrix belongs to dom(Ric) and that $X = \mathrm{Ric}(H) \geqq 0$.

$$H = \begin{bmatrix} A & 0 \\ -C1^*C1 & -A^* \end{bmatrix} - \begin{bmatrix} B2 \\ -C1^* D12 \end{bmatrix} \begin{bmatrix} D12^* C1 & B2^* \end{bmatrix}$$

$$= \begin{bmatrix} A - B2\, D12^* C1 & -B2\, B2^* \\ -C1^* D_\perp D_\perp^* C1 & -(A - B2\, D12^* C1)^* \end{bmatrix} \quad \text{---- (13.8)}$$

Note also that if $D12^* C1 = 0$, then A4 is implied by the detectability of (C1,A).

 # *********Corollary 12.7*****************

Suppose D has full column rank and denote $R = D^* D > 0$. Let H has the form of

$$H = \begin{bmatrix} A & 0 \\ -C^*C & -A^* \end{bmatrix} - \begin{bmatrix} B \\ -C^*D \end{bmatrix} R^{-1} \begin{bmatrix} D^*C & B^* \end{bmatrix}$$

$$= \begin{bmatrix} A - B\, R^{-1} D^*C & -B\, R^{-1} B^* \\ -C^* (I - D\, R^{-1} D^*)\, C & -(A - B R^{-1} D^* C)^* \end{bmatrix}$$

Then $H \in \mathrm{dom}(\mathrm{Ric})$ iff (A,B) is stabilizable and

$$\begin{bmatrix} A - j\omega I & B \\ C & D \end{bmatrix}$$

has full column rank for all ω. Further more, $X = \mathrm{Ric}(H) \geqq 0$ if $H \in \mathrm{dom}(\mathrm{Ric})$ and $\mathrm{Ker}(X) = 0$ if and only if ($D^*{}_\perp C1$, $A - BR^{-1}D^*C$) has no stable unobservable modes.

****

Note that the Riccati equation corresponding to equation (13.8) is

$$(A - B2\, d12^*\, C1)^* X + X(A - B2\, D12^*\, C1) - X\, b2\, B2^* X + C1^*\, D_\perp D_\perp^*\, C1 = 0 \quad \text{--- (13.9)}$$

Let X be the corresponding stabilizing solution and define

$$F = -(B2^* X + d12^* C1) \quad \text{---(13.10)}$$

Then, A + B2F is stable. denote

$$AF = A + B2\,F, \quad CF = C1 + d12\,F$$

and rearrange equation (13.9) to get

$$AF^* X + X\,AF + CF^*\,CF = 0 \quad \text{-- (,13.11)}$$

Thus X is the observability Gramian of (CF, AF).

%%%%%%%%%%%%%%%%%%%%%%

Consider applying the control law

u = F x to the system equation (13.6) and (13.7). The controlled system becomes

$$x' = AF\,x, \quad x(t=0) = xo \quad \text{and} \quad z = CF\,x$$

The associated transfer matrix is

$$Gc(s) = \begin{bmatrix} AF & I \\ CF & 0 \end{bmatrix}$$

and

$$\| Gc\, xo \|_2^2 = xo^*\, X\, xo$$

The proof of the following theorem needs a preliminary result about internal stability given input-out put stability.

Lemma 13.1 If $u,z \in L2[0, \infty)$ and (C1,A) is detectable in the system described by (13.6) and (13.7) then $x \in L2[0,\infty)$. In addition x(t) --> as t---> oo.

Proof. Since (C1, A) is detectable, there exists L such that A + LC1 is stable. let x^ be the state estimate of x

$$x' = (A + L\,C1)\,\hat{x} + (L\,d12 + B2)\,u - Lz.$$

Then, $\hat{x} \in L2[0,\infty)$ since z and u are in L2[0,oo). Now let e = x - x^. then

$$e' = (A + L\,C1)\,e$$

and $e \in L2[0,\infty)$. Therefore $x = e + \hat{x} \in L2[0,\infty)$.

e(t) -->0 as t-->oo.

Theorem 13.2 There exists a unique optimal control for the LQR problem, namely u = F x. Moreover

$$\min_{u \in L2[0,\infty)} \| z \|_2 = \| Gc\, xo \|_2$$

Note that the optimal control strategy is a constant gain state feed back which gain is independent of the initial x0.

Proof. With the change of variable v = u - Fx, the system can be written as

$$\begin{bmatrix} x' \\ z \end{bmatrix} = \begin{bmatrix} AF & B2 \\ CF & D12 \end{bmatrix} \begin{bmatrix} x \\ v \end{bmatrix}, \quad x(0) = xo \quad \text{--(13.12)}$$

Now if $v \in L2[0,\infty)$ and $x(\infty) = 0$ since AF is stable. Hence $u = Fx + v \in L2[0,\infty)$. Conversely if $u,z \in L2[0,\infty)$, then from the Lemma13.1 $x \in l2[0,\infty)$. So $v \in L2[0,\infty)$. Thus mapping v = u - Fx between $v \in L2[0,\infty)$ and those $u \in L2[0,\infty)$ that make $z \in L2[0,\infty)$ is one to one and onto. therefore

$$\min_{u \in L2[0,\infty)} \| z \|_2 = \min_{v \in L2[0,\infty)} \| z \|_2$$

By differentiating x(t)* X x(t) with respect to t along a solution of the differential equation (13.12) and by using equation (13.9) and the fact that CF* D12 = - X B2,

$$\begin{aligned} \partial(x^* X x)/\partial t &= x^{*\prime} X x + x^* X x' \\ &= x^* (AF\,X + X\,AF)\,x + 2\,x^* X B2\, v \\ &= -x^* CF^*\,CF\,x + 2\,x^* X B2\, v \\ &= -(CF\,x + D12\,v)^* (CF\,x + D12\,v) \\ &\quad + 2\,x^* CF^* D12\, v + v^* v + 2\,x^* X B2\, v \\ &= -\| z \|_2^2 + \| v \|_2^2 \quad \text{----------(13.13)} \end{aligned}$$

by integrating (13.13) from 0 to oo.

$$\| z \|_2^2 = xo^*\, X\, xo + \| v \|_2^2$$

Clearly the unique optimal control is v =0,

u = F x.

3. Guaranteed stability margins of LQR.

We consider the system of

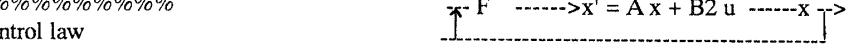
$$x' = A\,x + B2\,u \quad \text{-----(13-6)}$$

$$z = C1\,x + D12\,u \quad \text{------(13-7)}$$

with the LQR control law

u = F x.

The closed loop block diagram is



Lemma 13.4

Let F= - (B2* X + D12*C1) and define

$$G12 = D12 + C1\,(s\,I - A)^{-1} B2$$

Then,

$$(I - B2^*\,(-s\,I - A^*)^{-1} F^*)\,(I - F\,(s\,I - A)^{-1} B2) = \tilde{G12}(s)\, G12(s)$$

Proof. Note that the Riccati equation (13.9) can be written as

$$XA + A^*X - F^* F + C1^*\, C1 = 0$$

Add and subtract s X to the above equation

$$-X\,(s\,I - A) - (-s\,I - A^*)\,X - F\,F^* + C1^*\, C1 = 0$$

Now multiplying the above equation from the left by

$$B2^*\,(-s\,I - A^*)^{-1}$$

and from the right by $(s\,I - A)^{-1}$ we have

$$\begin{aligned} &-B2^*\,(-s\,I - A^*)^{-1}\, X\, B2 - B2^*\, X\,(s\,I - A)^{-1} B2 \\ &- B2^*\,(-sI - A^*)^{-1} F^*\, F\,(s\,I - A)^{-1} B2 \\ &+ B2^*\,(-s\,I - A^*)^{-1} C1^*\, C1\,(s\,I - A)^{-1}\, B2 = 0 \end{aligned}$$

Using $-B2^*X = F + D12^*\, C1$ in the above equation,

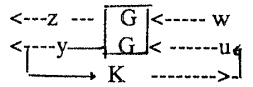
$$\begin{aligned} &B2^*\,(-s\,I - A^*)^{-1}\, F^* + F\,(s\,I - A)^{-1} B2 \\ &- B2^*\,(-sI - A^*)^{-1} F^*\, F\,(s\,I - A)^{-1} B2 \\ &+ B2^*\,(-s\,I - A^*)^{-1} C1^* D12 + D12^* C1\,(s\,I - A)^{-1}\, B2 \\ &+ B2^*\,(-s\,I - A^*)^{-1} C1^*\, C1\,(s\,I - A)^{-1}\, B2 = 0 \end{aligned}$$

Because D12* D12 =I, we have

$$(I - B2^*\,(-s\,I - A^*)^{-1} F^*)\,(I - F\,(s\,I - A)^{-1} B2) = \tilde{G12}(s)\, G12(s)$$

13.5 Standard H2 problem.

The system considered is



The realization of the transfer matrix G is taken to be

$$G(s) = \begin{bmatrix} A & B1 & B2 \\ C1 & 0 & D12 \\ C2 & D21 & 0 \end{bmatrix}$$

The special off-diagonal structure of D. D22 is assumed to be zero so that G22 is strict;y proper also d11 us assumed to be zero in order to guarantee that h2 problem is properly posed. The following additional assumptions were made for the output feed back H2 problem.

1. (A, B2) is stabilizable and (C2. A) is detectable.

This assumption is for the stabilizability of G by output fed back.

2. R1 = D12* D12 >0 and R2 = D21 D21* > 0.

This assumption guarantees that the H2 optimal control problem is non singular.

3. $\begin{bmatrix} A - j\omega I & B2 \\ C1 & D12 \end{bmatrix}$ **has full column rank for all** ω.

4. $\begin{bmatrix} A - j\omega I & B1 \\ C2 & D21 \end{bmatrix}$ **has full row rank for all** ω.

The third and fourth assumptions together with the first ones guarantee that the two Hamiltonian matrices associated with the following H2 problem belong to dom (Ric).

&&& * H2 Problem *** &&&**

The H2 control problem is to find a proper real rational controller K that stabilizes G internally and minimizes the H2 norm of the transfer matrix Tzw from w.

In the following, we assume that we have state models of G and K. Recall that a controller is said to be admissible if it is internally stabilizing and proper. By **Corollary 12.7** the two Hamiltonian matrices

$$H2 = \begin{bmatrix} A - B2\,R1^{-1}\,D12^*\,C1 & -B2R1^{-1}B2^* \\ -C1^*\,(I - D12\,R1^{-1}\,D12^*)\,C1 & -(A - B2\,R1^{-1}\,D12^*\,C1)^* \end{bmatrix}$$

$$J2 = \begin{bmatrix} (A - B1D21^*\,R2^{-1}\,C2 & -C2^*R2^{-1}C2 \\ -B1\,(I - D21^*\,R2^{-1}\,D21)\,B1^* & -(A - B1\,D21^*R2^{-1}\,C2) \end{bmatrix}$$

belong to dom(Ric) and

$X2 = Ric\,(H2) \geqq 0$ and $Y = Ric\,(J2) \geqq 0$.

Define

$F2 = -R1^{-1}\,(B2^*\,X2 + D12^*\,C1)$

$L2 = -(Y2\,C2^* + B1\,D21^*)\,R2^{-1}$ and

$AF2 = A + B2\,F2$, $C1F2 = C1 + D12\,F2$

$AL2 = A + L2\,c2$ $B1L2 = B1 + L2\,D21$

$$Gc(s) = \left[\begin{array}{c|c} AF2 & I \\ \hline C1F2 & 0 \end{array}\right] \qquad Gf(s) = \left[\begin{array}{c|c} AL2 & B1L2 \\ \hline I & 0 \end{array}\right]$$

Lemma 13.6 Let U and V $\in$ RHoo be defined as

$$U = \begin{bmatrix} AF2 & B2\,R1^{-1/2} \\ C1F2 & D12\,R1^{-1/2} \\ AL2 & B1L2 \end{bmatrix}$$

$$V = \begin{bmatrix} R2^{-1/2}\,C2 & R2^{-1/2}\,D21 \end{bmatrix}$$

Then U is an inner and V is a co-inner

$U\sim Gc \in RH2^{\perp}$

$Gf\,V\sim \in RH2^{\perp}$

Proof. The proof utilizes the standard manipulation of state space realization. From U. we get

$$U^T(-s) = U\sim(s) = \begin{bmatrix} -AF2^* & -C1^*F2 \\ R1^{-1/2}\,B2^* & R1^{-1/2}\,D12^* \end{bmatrix}$$

Then,

$$U\sim U = \begin{bmatrix} -AF2^* & -C1^*F2\,C1F2 & -C1^*F2\,D12\,R1^{-1/2} \\ 0 & AF2 & B2\,R1^{-1/2} \\ R1^{-1/2}\,B2^* & R1^{-1/2}\,D12^*\,C1F2 & I \end{bmatrix}$$

$$U\sim Gc = \begin{bmatrix} -AF2^* & -C1^*F2\,C1F2 & 0 \\ 0 & AF2 & I \\ R1^{-1/2}\,B2^* & R1^{-1/2}\,D12^*\,C1F2 & 0 \end{bmatrix}$$

By the similarity transformation

$$\begin{bmatrix} I & -X2 \\ 0 & I \end{bmatrix}$$

on the states of the proceeding transfer matrixes and note that

$AF2^*\,X2 + X2\,AF2 + C1^*F2\,C1F2 = 0$

we have

$$U\sim U = \begin{bmatrix} -AF2^* & 0 & 0 \\ 0 & AF2 & B2\,R1^{-1/2} \\ R1^{-1/2}\,B2^* & 0 & I \end{bmatrix} = I$$

$$U\sim Gc = \begin{bmatrix} -AF2^* & 0 & -X2 \\ 0 & AF2 & I \\ R1^{-1/2}\,B2^* & 0 & 0 \end{bmatrix} =$$

$$= \begin{bmatrix} -AF2^* & -X2 \\ R1^{-1/2}\,B2^* & 0 \end{bmatrix} \in RH2^{\perp}$$

Theorem 13. 7

There exist a unique optimal controller

$$Kopt(s) = \left[\begin{array}{c|c} A2^{\wedge} & -L2 \\ \hline F2 & 0 \end{array}\right]$$

and

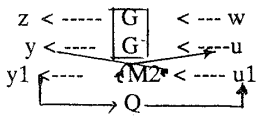
$$\min \|Tzw\|_2^2 = \|Gc\,B1\|_2^2 + \|R1^{-1/2}\,F2\,Gf\|_2^2 = trace\,(B1^*X2b1) + trace\,(r1\,F2\,Y2\,F2^*)$$

Proof. Consider the all stabilizing controller parameterization

$K(s) = FL\,(M2, Q)$, $Q \in RHoo$ with

$$M2(s) = \left[\begin{array}{cc|c} A2^{\wedge} & -L2 & B2 \\ F2 & 0 & I \\ \hline -C2 & I & 0 \end{array}\right]$$

and consider the following diagram



Then $Tzw = FL\,(N, Q)$ with

$$N = \begin{bmatrix} AF2 & -B2\,F2 & B1 & B2 \\ 0 & AL2 & B1\,L2 & 0 \\ C1F2 & -D12\,F2 & 0 & D12 \\ 0 & C2 & D21 & 0 \end{bmatrix}$$

and

$Tzw = Gc\,B1 - U\,R1^{1/2}\,F2\,Gf + U\,R1^{1/2}\,Q\,R2^{1/2}\,V$

It follows from Lemma 13.6 that GcB1 and U are orthogonal. Thus

$$\|Tzw\|_2^2 = \|Gc\,B1\|_2^2 + \|U\,R1^{1/2}\,F2\,Gf - U\,R1^{1/2}\,Q\,R2^{1/2}\,V\|_2^2 = \|Gc\,B1\|_2^2 + \|R1^{1/2}\,F2\,Gf - R1^{1/2}\,Q\,R2^{1/2}\,V\|_2^2$$

Since Gf and V are also orthogonal by Lemma 13.6

$$\|Tzw\|_2^2 = \|Gc\,B1\|_2^2 + \|R1^{1/2}\,F2\,Gf - R1^{1/2}\,Q\,R2^{1/2}\,V\|_2^2 = \|Gc\,B1\|_2^2 + \|R1^{1/2}\,F2\,Gf\|_2^2 + \|R1^{1/2}\,Q\,R2^{1/2}\|_2^2$$