Calcium Stimulates Intramitochondrial Cholesterol Transfer in Bovine Adrenal Glomerulosa Cells*

Nadia Cherradi‡, Michel F. Rossier§, Michel B. Vallotton, and Alessandro M. Capponi

From the Division of Endocrinology and Diabetology, Department of Medicine, Faculty of Medicine, CH-1211 Geneva, 14 Switzerland

In adrenal glomerulosa cells, angiotensin II (Ang II) stimulates aldosterone synthesis through rises of cytosolic calcium ([Ca\(^{2+}\)]\(_c\)). The rate-limiting step in this process is the transfer of cholesterol to the inner mitochondrial membrane, where it is converted to pregnenolone by the P450 side chain cleavage enzyme. The aim of the present study was to examine the effect of changes in [Ca\(^{2+}\)]\(_c\), and of Ang II on intramitochondrial cholesterol distribution. Freshly prepared bovine zona glomerulosa cells were submitted to a cytosolic Ca\(^{2+}\) clamp (600 nM) or stimulated with Ang II (10 nM). Mitochondria were isolated and subfractionated into outer membranes (OM), inner membranes (IM), and contact sites (CS). Cholesterol content was determined by the cholesterol oxidase assay. Stimulation of intact cells with Ca\(^{2+}\) led to a marked decrease in cholesterol content of OM (to 54 ± 24% of controls, n = 5) and to a concomitant increase of cholesterol in CS and IM (to 145 ± 14%, n = 5). When glomerulosa cells were exposed to Ang II, a marked increase of cholesterol in CS occurred (to 172 ± 16% of controls, n = 5). No significant changes were detected in OM cholesterol, suggesting a stimulation of cholesterol supply to the mitochondria in response to Ang II. Cycloheximide specifically and significantly reduced Ca\(^{2+}\)-activated cholesterol transfer to CS and IM. In conclusion, our data indicate that one of the main functions of the Ca\(^{2+}\) messenger is to increase cholesterol supply to the P450 side chain cleavage enzyme by enhancing endogenous intermembrane cholesterol transfer to a mitochondrial site containing the enzymes responsible for the initial steps of the steroidogenic cascade.

The major physiological regulators of aldosterone synthesis and secretion by adrenal glomerulosa cells are angiotensin II (Ang II)\(^1\) and potassium (1). Although acting through different mechanisms, these two extracellular stimulatory factors trigger steroidogenesis by a process involving the calcium messenger system (2–10).

The crucial role of the Ca\(^{2+}\) messenger in the acute regulation of aldosterone production in adrenal glomerulosa cells is well recognized (4, 11, 12). However, the sites of action and the specific molecular targets of calcium along the complex steroidogenic cascade are poorly defined. A first direct indication of the involvement of the adrenal glomerulosa mitochondria as targets for the Ca\(^{2+}\) messenger has been obtained when our laboratory has shown using permeabilized bovine glomerulosa cells that changes of ambient Ca\(^{2+}\) within the range of the physiological cytosolic concentrations are able to activate aldosterone production and that this effect can be prevented by ruthenium red, a blocker of the mitochondrial Ca\(^{2+}\) uniport (13). Moreover, recent work has allowed us to narrow the potential target domain for Ca\(^{2+}\) to the very early steps of steroidogenesis (14), which occur inside the mitochondria.

Indeed, the acute response of steroidogenic cells to hormone stimulation involves the mobilization of cholesterol from intracellular lipid droplets to the mitochondrial inner membrane, where the first enzymatic step of steroidogenesis, namely the conversion of cholesterol to pregnenolone by the cytochrome P450\(_{acx}\) occurs (15). The rate-limiting and hormonally regulated step in this process is the delivery of cholesterol from the outer to the inner mitochondrial membrane. This step is known to require de novo protein synthesis. Studies from several laboratories have shown that a family of hormone-induced and cycloheximide-sensitive 30-kDa mitochondrial proteins, described in different steroidogenic cell types (16–20), play a crucial role in the acute regulation of steroid synthesis. Recently, the steroidogenic acute regulatory (STAR) protein has been proposed as an essential mediator of the acute steroidogenic response. Cholesterol transfer is believed to be facilitated by contact sites that occur between the outer and the inner mitochondrial membranes during the import of the STAR protein precursor into the mitochondria (20). This hypothesis is strengthened by many observations showing that mitochondrial contact sites are involved in phospholipid and protein import into the mitochondria (21, 22).

Although the regulation of the cholesterol transfer steps has been almost exclusively investigated in response to elevated cAMP levels in adrenal fasciculata cells, many questions remain unanswered concerning the possible role of Ca\(^{2+}\) in the regulation of cholesterol mobilization in adrenal glomerulosa cells. The aims of the present study were firstly to determine whether the changes in intracellular calcium concentration triggered by activators of steroidogenesis in glomerulosa cells are accompanied by concomitant changes in cholesterol distribution in mitochondrial membranes and secondly to examine the effect of the inhibitor of protein translation, cycloheximide (CHX), on the calcium-mediated cholesterol transfer from the outer to the inner mitochondrial membrane.

---

* This work was supported by Swiss National Science Foundation Grants 31.42178-94 (to A. M. C.) and 32.39277-93 (to M. F. R.). The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

‡ To whom correspondence should be addressed: Division of Endocrinology and Diabetology, University Hospital, 24, Micheli-du-Crest, CH-1211 Geneva 14, Switzerland. Tel.: 4122-372-93-21; Fax: 4122-372-93-29.

§ Recipient of a grant from the Prof. Max Clotetta Foundation.

\(^1\) The abbreviations used are: Ang II, angiotensin II; [Ca\(^{2+}\)]\(_c\), cytosolic free calcium concentration; P450\(_{acx}\), cytochrome P450 side chain cleavage enzyme; AMG, aminolucetamide; CHX, cycloheximide; NDP, nucleoside-diphosphate; ACTH, adrenocorticotropic hormone.
**EXPERIMENTAL PROCEDURES**

**Materials**—Ionomycin was purchased from Calbiochem (Laurenc, Switzerland) and [Ile]3-Ang II from Bachem (Bubendorf, Switzerland). Cholesterol oxidase, peroxidase, p-hydroxyphenylacetic acid, aminoglutethimide, cycloheximide, and all other chemicals were obtained from Sigma.

**Bovine Adrenal Zona Glomerulosa Cell Preparation**—Bovine adrenal glands were obtained from a local slaughterhouse. Zona glomerulosa cells were prepared by enzymatic dispersion with dispase and purified on Percoll density gradients as described in detail elsewhere (23). Purified glomerulosa cells were resuspended in 10 mM sodium phosphate buffer, pH 7.4, containing 0.1% bovine serum albumin as a standard.

**Characterization of the Submitochondrial Fractions**—Membranes of osmotically lysed mitochondria isolated from glomerulosa cells in which [Ca2+]o had been previously clamped for 2 h at either low levels (500 nM) or high levels (600 nM) were separated into various fractions by continuous sucrose density gradient centrifugation as described under "Experimental Procedures." Submitochondrial particles were prepared on a 15–50% sucrose density gradient as described. The protein content (A) and the activities of mitochondrial marker enzymes (B) were determined in duplicate in each fraction of the gradient. The activity profiles are representative of eight independent experiments. MAO, monoamine oxidase; COX, cytochrome c oxidase; ATP, adenosine triphosphatase; MAO activity units are pmol deaminated tryptamine/min/mg protein for monoamine oxidase, nmol oxidized cytochrome c/min/mg protein for cytochrome c oxidase, and nmol ADP/min/mg protein for NDP kinase.

**RESULTS**

**Characterization of the Submitochondrial Fractions**—Membranes of osmotically lysed mitochondria isolated from glomerulosa cells in which [Ca2+]o had been previously clamped for 2 h at either low levels (<100 nM, control cells) or at high levels (600 nM) were separated into various fractions by continuous sucrose density gradient centrifugation as described under "Experimental Procedures." The protein distribution in the gradient under each condition is shown in Fig. 1A. Fig. 1B illustrates the separation of mitochondrial membranes from control (low [Ca2+]o) glomerulosa cells. The activity profile of specific mitochondrial marker enzymes in the gradient led to the characterization of three distinct membrane populations. A first population with the lowest density (fractions 5–8) showed the highest monoamine oxidase activity, which is specific of the outer mitochondrial membranes. The population of membranes with the highest density (fractions 13–15) exhibits the highest content of cytochrome c oxidase activity, as expected for the inner mitochondrial membranes. In addition, a third mem-

![Fig. 1. Separation of submitochondrial membranes by density gradient centrifugation. Bovine adrenal glomerulosa cells were submitted for 2 h to a Ca2+ clamp in the presence of 500 nM AMG as described under “Experimental Procedures.” Submitochondrial particles were prepared on a 15–50% sucrose density gradient as described. The protein content (A) and the activities of mitochondrial marker enzymes (B) were determined in duplicate in each fraction of the gradient. The activity profiles are representative of eight independent experiments. MAO, monoamine oxidase; COX, cytochrome c oxidase; ATP, adenosine triphosphatase; MAO activity units are pmol deaminated tryptamine/min/mg protein for monoamine oxidase, nmol oxidized cytochrome c/min/mg protein for cytochrome c oxidase, and nmol ADP/min/mg protein for NDP kinase.](http://www.jbc.org/)

**Fig. 1. Separation of submitochondrial membranes by density gradient centrifugation.** Bovine adrenal glomerulosa cells were submitted for 2 h to a Ca2+ clamp in the presence of 500 nM AMG as described under “Experimental Procedures.” Submitochondrial particles were prepared on a 15–50% sucrose density gradient as described. The protein content (A) and the activities of mitochondrial marker enzymes (B) were determined in duplicate in each fraction of the gradient. The activity profiles are representative of eight independent experiments. MAO, monoamine oxidase; COX, cytochrome c oxidase; ATP, adenosine triphosphatase; MAO activity units are pmol deaminated tryptamine/min/mg protein for monoamine oxidase, nmol oxidized cytochrome c/min/mg protein for cytochrome c oxidase, and nmol ADP/min/mg protein for NDP kinase.
The cholesterol content of each submitochondrial fraction of control or high Ca\(^{2+}\)-clamped cells was determined by a cholesterol oxidase-peroxidase assay as described under “Experimental Procedures.” The cholesterol content in subunitochondrial fractions of high Ca\(^{2+}\)-clamped cells is expressed as a percentage of that measured in submitochondrial fractions of control cells (mean ± S.E., n = 5). * and **, significantly different from the respective control with p < 0.05, p < 0.01, and p < 0.001, respectively. In the inset, the cholesterol content of each fraction is expressed as a function of total mitochondrial cholesterol content.

FIG. 2. Effect of a cytosolic Ca\(^{2+}\) clamp on cholesterol content of submitochondrial fractions of bovine glomerulosa cells. The cholesterol content of each submitochondrial fraction of control or high Ca\(^{2+}\)-clamped cells was determined by a cholesterol oxidase-peroxidase assay as described under “Experimental Procedures.” The cholesterol content in submitochondrial fractions of high Ca\(^{2+}\)-clamped cells is expressed as a percentage of that measured in submitochondrial fractions of control cells (mean ± S.E., n = 5). *, **, and ***, significantly different from the respective control with p < 0.05, p < 0.01, and p < 0.001, respectively. In the inset, the cholesterol content of each fraction is expressed as a function of total mitochondrial cholesterol content.

Cholesterol content (% of control)

Fraction

FIG. 3. Effect of CHX on Ca\(^{2+}\)-induced cholesterol transfer in adrenal glomerulosa mitochondria. The effect of Ca\(^{2+}\) on mitochondrial endogenous cholesterol transfer was assessed as described in the legend of Fig. 2, except that in one lot of glomerulosa cells, 1 μM CHX was added to the incubation medium 30 min before the Ca\(^{2+}\) clamp. Each point is the mean ± S.E. of four independent cell preparations. *, **, and ***, significantly different from the respective control, with p < 0.05, p < 0.01, and p < 0.001, respectively. * and **, significantly different from the corresponding value obtained with Ca\(^{2+}\) alone, with p < 0.05 and p < 0.01, respectively.

Calculated Is a Potent Stimulator of Intramitochondrial Cholesterol Transfer—We have previously shown in bovine adrenal zona glomerulosa cells that the calcium-ionophore, ionomycin, can be effectively used at low concentration to clamp the cytosolic free Ca\(^{2+}\) concentration, [Ca\(^{2+}\)]\(_{c}\), at various physiological levels (50–1000 nM) (14). This submicromolar [Ca\(^{2+}\)]\(_{c}\) stimulates the early mitochondrial steps of steroidogenesis, namely pregnenolone formation from cholesterol side chain cleavage, as well as aldosterone synthesis (14).

Cholesterol content was determined in the various fractions of the sucrose gradient. In Fig. 2, the total cholesterol content in each submitochondrial fraction from high Ca\(^{2+}\)-clamped cells has been expressed as a percentage of the cholesterol content measured in the corresponding submitochondrial fractions from control cells. The stimulation of intact glomerulosa cells with Ca\(^{2+}\) led to a marked decrease of cholesterol content in the outer mitochondrial membranes (fraction 5, corresponding to the peak of monoamine oxidase activity: 54 ± 24% of controls, n = 5) with a concomitant increase in contact sites (fraction 11, corresponding to the peak of NDP kinase activity: 145 ± 14% of controls) and a less pronounced augmentation in the inner mitochondrial membranes (fraction 14, corresponding to the peak of cytochrome c oxidase activity: 125 ± 5% of controls).

These results demonstrate that Ca\(^{2+}\) activates mitochondrial endogenous cholesterol transfer from the outer membrane to the contact sites and inner membrane, a process that is accompanied by an increased aldosterone production. Indeed, the aldosterone output measured in Ca\(^{2+}\)-clamped cells incubated in the absence of AMG amounted to 345 ± 42% of controls (n = 8).

Ca\(^{2+}\)-stimulated Cholesterol Transfer from the Outer to the Inner Membrane Is Sensitive to Cycloheximide—Earlier reports have indicated that ACTH-activated cholesterol transport to the mitochondrial inner membrane of steroidogenic cells is blocked by inhibitors of protein synthesis such as CHX, with a resulting ACTH-induced accumulation of cholesterol in the outer mitochondrial membrane (15, 28). Moreover, it has been reported that the increase in pregnenolone and aldosterone synthesis triggered by Ang II is inhibited by the same compound (29), suggesting that at the mitochondrial level, both hormones operate through similar mechanisms. We have therefore tested whether CHX may also inhibit Ca\(^{2+}\)-stimulated intramitochondrial cholesterol transfer in glomerulosa cells. Fig. 3 shows that when CHX was added concomitantly with Ca\(^{2+}\), the outer membrane fractions 5 and 6 contained 82 ± 4.4 and 82 ± 4.9% of the cholesterol content of their respective control fractions, as compared with only 68.3 ± 4.8% and 67 ± 3.6%, respectively, when the Ca\(^{2+}\) clamp was performed in the absence of CHX (p < 0.05, n = 4). By contrast, CHX significantly reduced Ca\(^{2+}\)-activated cholesterol transfer to contact sites (fractions 11–12) and inner membranes (fractions 13–14) (Fig. 3). Cycloheximide similarly prevented Ang II-induced cholesterol transfer to contact sites and inner membranes (data not shown).

Ang II Stimulates Exogenous Cholesterol Transport to Mitochondria, with a Concomitant Transfer of Endogenous Cholesterol to Contact Sites—In order to test whether Ang II-mediated cholesterol mobilization in mitochondria is similar to that triggered by the cytosolic Ca\(^{2+}\) clamp, glomerulosa cells were incubated for 2 h in the presence of 10 nM Ang II and 500 μM AMG. Submitochondrial membranes were prepared and analyzed as above. No change in protein profile of sucrose gradients could be observed upon Ang II stimulation (data not shown). Fig. 4 illustrates the distribution of cholesterol content...
in the sub mitochondrial fractions of glomerulosa cells exposed to Ang II. Firstly, the hormone induced a pronounced increase of cholesterol content in the contact site-enriched fractions (fractions 11 and 12, 172 ± 16 and 169 ± 6% of controls, respectively, n = 5). In separate experiments, we have observed a significant (28 ± 5%, n = 5) increase in total mitochondrial cholesterol of Ang II-stimulated-cells. Secondly, no significant changes were detected in the outer mitochondrial membranes (fractions 5 and 6, 83 ± 6 and 80 ± 11% of controls, respectively, n = 5). Thirdly, no increase in cholesterol content was observed in the inner membrane fractions (fractions 13 and 14, 109 ± 13 and 76 ± 22% of controls, respectively, n = 5). Interestingly, a subpopulation of the inner membranes revealed a significant decrease in cholesterol content, as compared with the respective control fractions (fractions 15, 16, and 17, 64 ± 4, 52 ± 6, and 57 ± 13% of controls, respectively, n = 5).

**DISCUSSION**

In the present study, we took advantage of the cytosolic Ca\(^{2+}\) clamp technique to investigate the Ca\(^{2+}\) sensitivity of intramitochondrial cholesterol transfer in bovine glomerulosa cells. Recently, using the ionomycin-mediated Ca\(^{2+}\) clamp, our laboratory has provided a first direct demonstration that [Ca\(^{2+}\)]\(_i\) in the submicromolar range stimulates aldosterone synthesis in intact glomerulosa cells (14). Because Ca\(^{2+}\) affects the formation of pregnenolone, an early mitochondrial step of aldosterone production (14), one could expect that or several target(s) of the Ca\(^{2+}\) messenger are located within the mitochondria.

The overall rate-limiting step of the steroidogenic cascade is the transport of cholesterol from a presteroidogenic pool in the outer membrane to a steroidogenic pool in the inner membrane (15, 28, 30). The regulation by ACTH of cholesterol distribution in adrenal mitochondria has been studied by several groups using mitochondrial disruption to yield outer and inner membranes (28, 31, 32). The hormone has been shown to activate cholesterol supply to the P450\(_{sec}\) enzyme in the inner membrane. However, restricting the separation to two major fractions may fail to uncover additional, functionally relevant membranous structures. We therefore fractionated bovine adrenal glomerulosa mitochondria into outer membranes, inner membranes, and intermembrane contact sites. The latter fraction contains marker enzymes for both membranes, in addition to NDP kinase activity (Fig. 1), which has shown to be specific of mitochondrial contact sites in several tissues (33).

The data presented here show that endogenous cholesterol transfer from the outer to the inner mitochondrial membrane and contact sites is substantially stimulated by physiological levels of cytosolic Ca\(^{2+}\). The supply of cholesterol to the P450\(_{sec}\) appears thus to be a Ca\(^{2+}\)-sensitive step in the early steroidogenic pathway. This observation is in agreement with the data recently reported by Kowluru and colleagues (34), showing that Ca\(^{2+}\) stimulates the metabolism of endogenous cholesterol to pregnenolone in rat adrenal mitochondria. Our previous studies have demonstrated that the P450\(_{sec}\) enzyme is located in the contact sites, in addition to being in the inner membrane of bovine adrenocortical mitochondria (24). Interestingly, the present data indicate that the Ca\(^{2+}\)-induced increase in cholesterol content is greater in the contact sites than in the inner membrane (Fig. 2). One interpretation could be that the P450\(_{sec}\) located in the contact sites is more active than the P450\(_{sec}\) of the inner membrane. Indeed, contact sites are known to be enriched in cardiolipin, a phospholipid that enhances the affinity of the P450\(_{sec}\) for cholesterol (35), although other explanations may be envisaged. Moreover, our results are in agreement with the data reported by Stevens et al. (36), suggesting that in addition to the outer and inner membrane pools, a third pool of steroidogenic cholesterol may be found in contact sites. On the other hand, Ca\(^{2+}\) induces direct contacts between the outer and the inner mitochondrial membranes, presumably by promoting a nonbilayer configuration leading to membrane fusion (37–39). Such an increase in the number of contact sites could result in an increase in the rate of cholesterol transfer.

Recently, Clark and colleagues (40) have observed that the elevations of cytosolic calcium triggered by Ang II, K\(^+\), and the Ca\(^{2+}\) channel agonist, BayK8644, are accompanied by increases in the level of the 30-kDa STAProtein in the human H295R adrenocarcinoma cell line. STAProtein import into the mitochondria via contact sites is thought to be a crucial event promoting cholesterol transfer to the inner membrane (20). We have also observed an increase in STAProtein content of mitochondria isolated from Ca\(^{2+}\)-clamped bovine glomerulosa cells.2 Our results therefore lead us to conclude that the STAProtein participates in the Ca\(^{2+}\)-induced cholesterol transfer in bovine glomerulosa mitochondria.

The above hypothesis is strengthened by the sensitivity of Ca\(^{2+}\)-induced cholesterol transfer to cycloheximide, an inhibitor of protein translation that depletes a set of 30-kDa proteins involved in the activation of steroidogenesis (16). From our study, it appears that CHX partially inhibits Ca\(^{2+}\)-stimulated cholesterol transfer from the outer membrane to the contact sites and inner membranes, suggesting that at least one part of the calcium-activated cholesterol transfer process requires protein synthesis. This finding is in agreement with the results reported by Kowluru et al. (34), suggesting that two intramitochondrial cholesterol transfer processes are mediated by Ca\(^{2+}\). One of these mechanisms involves mitochondrial membrane sites accessible to activation by Ca\(^{2+}\) and GTP (presumably the sites of STAP-mediated cholesterol transfer). In the presence of CHX, this process is blocked, leading to redistribution of cholesterol to other sites involving mitochondrial membrane contact sites resulting from Ca\(^{2+}\)-induced matrix swelling.

Several conclusions can be drawn from the experiments with

---

2 N. Cherradi, R. Tinberg, I. Friedberg, J. Orly, X. Wang, C. Stocco, and A. Capponi, submitted for publication.
Ang II stimulation. The lack of a decrease of cholesterol content in the outer membranes can be explained by a stimulation of cholesterol supply to the mitochondria in response to Ang II. Furthermore, a striking feature of the present study was the marked increase of cholesterol content in the contact sites, as compared with inner membranes, when glomerulosa cells were challenged with Ang II. This finding suggests that the hormone markedly enhances cholesterol availability in regions where the intermembrane space barrier is abolished. Interestingly, a subpopulation of the inner membranes even showed a decrease of cholesterol content, suggesting either a possible diffusion of cholesterol to specific sites in the inner membrane or a more unlikely selective loss of cholesterol during the fractionation procedure of mitochondria. Although we have no clear explanation at the present time for this result, it is worth mentioning that contact sites may be preferential sites for cholesterol transfer and metabolism, because they are enriched in the phospholipid cardiolipin, which promotes nonbilayer structures and enhances the affinity of P450 \(_{22}\) for cholesterol (35).

One could therefore speculate that in addition to activating cholesterol transfer from the outer to the inner membrane, Ang II may also affect cholesterol movement within the inner membrane.

In conclusion, using the ionomycin-mediated cytosolic \(Ca^{2+}\) clamp, we have shown that \(Ca^{2+}\) itself is able to activate cholesterol transfer from the outer to the inner mitochondrial membrane and to intermembrane contact sites in bovine glomerulosa cells, a process that occurs even in the absence of cholesterol metabolism to pregnenolone. In fact, cholesterol flux could be even more important if steroidogenesis were allowed to proceed. We also demonstrate that Ang II, a \(Ca^{2+}\)-mobilizing hormone, markedly increases cholesterol content in contact sites. Our results strongly suggest that one of the main functions of the \(Ca^{2+}\) messenger under hormonal stimulation is to increase cholesterol supply to the P450\(_{22}\) enzyme by enhancing intermembrane cholesterol transfer, thus promoting the activation of the subsequent steroidogenic cascade.

Acknowledgments—We are grateful to Liliane Bockhorn, Walda Dimeck, Gisèle Dorenter, Marcella Klein, and María Lopez for excellent technical assistance.

REFERENCES

1. Müller, J. (1988) Regulation of Aldosterone Biosynthesis, 2nd Ed., Springer-Verlag, Berlin.
2. Capponi, A. M., Lew, P. D., Jornot, L., and Vallotton, M. B. (1984) \(J\). Biol. Chem. \textbf{259}, 8863–8869.
3. Quinn, S. J., Williams, G. H., and Tillotson, D. L. (1988) \(Am. J. Physiol.\) \textbf{255}, E488–E495.
4. Barrett, P. Q., Bollag, W. B., Isales, C. M., McCarthy, R. T., and Rasmussen, H. (1989) \(Endocrinology\) \textbf{120}, 496–520.
5. Spat, A., Balla, I., Balla, T., Crago, E. J., Hajnóczky, G., and Hunyady, L. (1989) \(J. Endocrinol.\) \textbf{122}, 361–370.
6. Loo, U., and Vallotton, M. B. (1988) \(J. Biol. Chem.\) \textbf{263}, 8047–8050.
7. Rossi, M. F., Krause, K.-H., Lew, P. D., Capponi, A. M., and Vallotton, M. B. (1987) \(J. Biol. Chem.\) \textbf{262}, 4053–4058.
8. Ambrus, C., and Catt, K. J. (1992) \(Endocrinology\) \textbf{131}, 408–414.
9. Burrell, M. M., Pytyn, C. P., Vallotton, M. B., Capponi, A. M., and Rossi, M. F. (1994) \(Endocrinology\) \textbf{135}, 751–758.
10. Barrett, P. Q., Isales, C. M., Bollag, W. B., and McCarthy, R. T. (1991) \(Am. J. Physiol.\) \textbf{261}, F706–F719.
11. Fukundji, J. L., Chow, R., and Catt, K. J. (1979) \(Endocrinology\) \textbf{105}, 327–333.
12. Spat, A., Eneydi, P., Hajnóczky, G., and Hunyady, L. (1991) \(Exp. Physiol.\) \textbf{76}, 859–885.
13. Capponi, A. M., Rossi, M. F., Davies, E., and Vallotton, M. B. (1988) \(J. Biol. Chem.\) \textbf{263}, 16113–16117.
14. Python, C. P., Laban, O. P., Rossi, M. F., Vallotton, M. B., and Capponi, A. M. (1995) \(Biochem. J.\) \textbf{305}, 569–576.
15. Jefcoate, C. R., McNamara, B. C., Armentano, A., and Yamazaki, T. (1992) \(J. Steroid Biochem. Mol. Biol.\) \textbf{43}, 751–767.
16. Epstein, L. F., and Orme-Johnson, N. R. (1991) \(J. Biol. Chem.\) \textbf{266}, 19739–19745.
17. Stocco, D. M. (1992) \(J. Steroid Biochem. Mol. Biol.\) \textbf{43}, 319–333.
18. Elliott, M. E., Goodfriend, T. L., and Jefcoate, C. R. (1993) \(Endocrinology\) \textbf{123}, 1669–1677.
19. Clark, B. J., Wells, J., King, S. R., and Stocco, D. M. (1994) \(J. Biol. Chem.\) \textbf{269}, 28314–28322.
20. King, S. R., Ronen-Puehrmann, T., Timberg, R., Clark, B. J., Ory, J., and Stocco, D. M. (1995) \(Endocrinology\) \textbf{136}, 5165–5176.
21. Ardaal, D., Garnier, F., Lerné, F., Simonot, C., Louisot, P., and Gateau-Roesch, O. (1993) \(J. Biol. Chem.\) \textbf{268}, 25983–25992.
22. Schweiger, M., Herzog, V., and Neupert, W. (1987) \(J. Cell. Biol.\) \textbf{105}, 235–246.
23. Python, C. P., Rossi, M. F., Vallotton, M. B., and Capponi, A. M. (1993) \(Endocrinology\) \textbf{132}, 1489–1496.
24. Cherni, N., Defaye, G., and Chambaz, E. M. (1994) \(Endocrinology\) \textbf{134}, 1358–1364.
25. Appelmann, F., Wattiaux, R., and De Duve, C. (1955) \(Biochem. J.\) \textbf{59}, 438–445.
26. Otuka, S., and Kobayashi, Y. (1964) \(Biochem. Pharmacol.\) \textbf{13}, 995–1006.
27. Gamble, W., Vaughan, M., Kruth, H. S., and Avigan, J. (1978) \(J. Lipid Res.\) \textbf{19}, 1068–1070.
28. Privat, C., McNamara, B. C., Dhariali, M., and Jefcoate, C. R. (1987) \(Mol. Cell. Endocrinol.\) \textbf{53}, 87–101.
29. Elliott, M. E., and Goodfriend, T. L. (1984) \(Biochem. Pharmacol.\) \textbf{33}, 1519–1524.
30. Cheng, B., Hsu, D. K., and Kimura, T. (1983) \(Lipids\) \textbf{18}, 577–584.
31. Cheng, B., and Kimura, T. (1983) \(Lipids\) \textbf{18}, 577–584.
32. Privat, C., Crivello, J. F., and Jefcoate, C. R. (1983) \(Proc. Natl. Acad. Sci. U.S.A.\) \textbf{80}, 702–706.
33. Adams, V., Bosch, W., Schlegel, J., Wallimann, T., and Bürdicka, D. (1989) \(Biochim. Biophys. Acta.\) \textbf{981}, 213–225.
34. Kowalczyk, R., Yamazaki, T., McNamara, B. C., and Jefcoate, C. R. (1995) \(Mol. Cell. Endocrinol.\) \textbf{107}, 181–188.
35. Lambeth, J. D. (1981) \(J. Biol. Chem.\) \textbf{256}, 4757–4762.
36. Stevens, V. L., Xu, T., and Lambeth, J. D. (1992) \(Endocrinology\) \textbf{130}, 1557–1561.
37. Ardaille, D., Gasnier, F., Lerme, F., Simonot, C., Louisot, P., and Gateau-Roesch, O. (1989) \(Exp. Physiol.\) \textbf{74}, 408–414.
38. Bakker, A., De Bie, M., Benaert, I., Ravingerova, T., Zieglerhofer, A., Van Belle, H., and Jacob, W. (1993) \(Exp. J. Cell Biol.\) \textbf{31}, 66–70.
39. Bakker, A., Benaert, I., De Bie, M., Ravingerova, T., Zieglerhofer, A., Van Belle, H., and Jacob, W. (1994) \(Biochim. Biophys. Acta.\) \textbf{1224}, 583–588.
40. Clark, B. J., Pezzi, V., Stocco, D. M., and Rainey, W. E. (1995) \(Mol. Cell. Endocrinol.\) \textbf{115}, 215–219.
Calcium Stimulates Intramitochondrial Cholesterol Transfer in Bovine Adrenal Glomerulosa Cells

Nadia Cherradi, Michel F. Rossier, Michel B. Vallotton and Alessandro M. Capponi

*J. Biol. Chem.* 1996, 271:25971-25975.
doi: 10.1074/jbc.271.42.25971

Access the most updated version of this article at http://www.jbc.org/content/271/42/25971

Alerts:
- When this article is cited
- When a correction for this article is posted

Click here to choose from all of JBC's e-mail alerts

This article cites 39 references, 14 of which can be accessed free at http://www.jbc.org/content/271/42/25971.full.html#ref-list-1