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Chemisorption of Water at High Temperatures on Kaolinite: Effect on Dehydroxylation

Abstract. *The dehydroxylation reaction of kaolinite in a vacuum at 425°C is halted by introducing a water vapor pressure of 47 mm-Hg, and is resumed when the vacuum is reestablished. The sample gains weight corresponding to an approximately monomolecular layer of water on the kaolinite surface. At the temperatures and vapor pressures involved, the sorption is considered to be a chemisorption process.*

For some years, evidence has accumulated that the dehydroxylation of the mineral kaolinite, $Al_2Si_2O_5(OH)_4$, which takes place at temperatures exceeding about 420°C, is markedly dependent on the ambient water vapor pressure, even at pressures of a few millimeters of mercury. This dependence is not due to any rehydroxylation of the mineral because this requires high water vapor pressures (*1*). When pressed discs of the mineral are heated at constant temperature under normal atmospheric conditions, the overall rate of dehydroxylation decreases progressively as the disc thickness increases (*2*); it appears that the water vapor generated by the reaction has a self-retarding effect. When a partially reacted disc is sectioned, the interior may be little reacted when the exterior layer is fully reacted, even though the entire sample has been held at the same temperature for the same time (*3*).

More precise information is obtained when samples are reacted slowly under controlled water vapor pressures (*4, 5, 6*). Results obtained by Toussaint *et al.* (*5*) and by Brindley *et al.* (*6*), the latter employing vapor pressures from less than 10^{-3} mm-Hg to 157 mm-Hg, suggest that the marked effect of the water vapor atmosphere arises from the sorption of water on the mineral surface, which effectively reduces the area for dehydroxylation. At the temperatures and vapor pressures involved, approximately 425° to 525°C and up to 150 mm-Hg pressure, the sorption must be regarded as a chemisorption process.

Experiments have been carried out which show directly the sorption of water on kaolinite under these conditions. The sorbed water is taken up rapidly and halts the dehydroxylation reaction, and when a vacuum is reestablished the sorbed water is released, and the dehydroxylation proceeds normally.

We used a silica spiral microbalance (*6*) in an enclosed system where the reaction temperature and the water vapor pressure were independently controlled. Reactions were followed gravimetrically on loosely compacted samples (~ 100 mg), and results are expressed as a percentage change in the weight of the sample dried at room temperature in a vacuum.

Typical curves are shown (Fig. 1) for samples maintained at 425° ± 2°C, partly in a vacuum at a pressure lower than 10^{-3} mm-Hg, and partly at a vapor pressure of 47 mm-Hg. Curve I shows a run in a vacuum for 210 minutes; at the end of this time the dehydroxylation reaction has become quite slow. Introducing the water vapor causes an almost instantaneous weight increase of about 0.85 percent; removal of the vapor restores the original weight. Curve II shows the same dehydroxylation process, but in vacuum for only 60 minutes at which time the reaction is still proceeding rapidly. Introducing the water vapor halts the reaction, and there is a weight increase of 1.30 percent, the weight remaining constant for 60 minutes while the vapor atmosphere is maintained. When the vacuum is restored, the reaction is resumed along a continuation indicated by the original curve, with a time displacement of about 80 minutes. Curve III shows a run in which the vapor was introduced immediately after the heated furnace was raised around the reaction vessel. Apart from a small initial reaction attributable to establishing steady conditions, there is no further dehydroxylation for 75 minutes, until the vacuum is applied, and then the reaction proceeds normally; curve III is practically parallel with curve I. Curve IV shows that when the water-vapor atmosphere is continued for 4 hours there is no reaction after the first small effect.

If the effective area of a sorbed H_2O molecule is taken as 10.8 Å^2, the weight of sorbed water shown by curves I and II, around 1.0 percent, corresponds to an area of about 36 m^2/g. The surface area of the initial kaolinite determined by the nitrogen adsorption method of Brunauer, Emmett, and Teller is 30 m^2/g. The correspondence of these areas points to the establishment of an approximately monomolecular layer of sorbed water on the kaolinite. Other experiments (*7*) indicate that the surface area of kaolinite is likely to be little changed by partial dehydroxylation at 425°C.

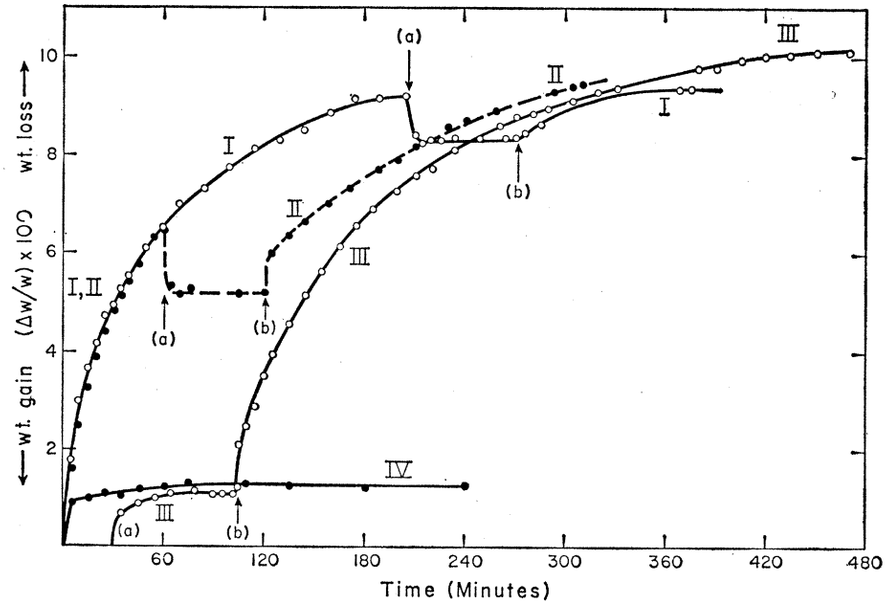

Fig. 1. Dehydroxylation of kaolinite at 425°C in a vacuum, or in a water-vapor atmosphere of 47 mm-Hg. Arrow (*a*) shows where water vapor is introduced, and (*b*) shows where the vacuum is restored.

Our results confirm the view that the rate of dehydroxylation of kaolinite (and probably of other similar hydroxyl-containing minerals) depends sensitively on vapor pressure because of chemisorption of water on the mineral surfaces.

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Puromycin Analogs: Action of Adrenocorticotropic Hormone and the Role of Glycogen

Abstract. *The effect of the injection into rats of analogs of puromycin, 6-dimethylaminopurine, and the aminonucleoside of puromycin on the stimulation of steroidogenesis by adrenocorticotropic hormone was compared with that of puromycin and cycloheximide. This stimulation was blocked only by the antibiotics, which also inhibited adrenal protein synthesis. Glycogenolysis is not associated with the primary mechanism of the adrenocorticotropic hormone stimulation of steroid hormone biosynthesis in rats.*

Adrenocorticotropic hormone (ACTH) stimulates steroid biosynthesis by a mechanism involving protein synthesis (*1–3*). The injection into rats of inhibitors of protein synthesis, puromycin or cycloheximide, blocked the ACTH stimulation of steroidogenesis in vivo. These studies suggested that this action of ACTH might be caused by activating the synthesis of protein undergoing rapid turnover (*3*). In that the injection of puromycin into mice resulted not only in an inhibition of protein synthesis, but also a depletion of hepatic glycogen (*4*), it has been suggested that the puromycin inhibition of ACTH-stimulated steroidogenesis could be explained primarily by its glycogenolytic effect (*5*).

We have attempted to clarify this issue and conclude that the antibiotics inhibited the ACTH stimulation by blocking protein synthesis.

Albino rats (200 g) were hypophysectomized from 2 to 6 hours before the start of the experiment. The effect of ACTH on corticosterone production by rat adrenals in vivo and the incorporation of radioactive amino acids into adrenal protein were determined as described (*3*). Glycogen concentration in liver and adrenal was determined by the Anthrone method (*6*).

The analogs of puromycin, 6-dimethylaminopurine, and the aminonucleoside of puromycin do not inhibit protein synthesis but, like puromycin, cause a decrease in hepatic glycogen concentration (*4*). To observe the effect of these compounds on the stimulation of steroidogenesis by ACTH, they were injected intraperitoneally into hypophysectomized rats; 15 minutes later there was a marked decrease in liver glycogen, but there was no change in adrenal glycogen (Table 1). However, the stimulation of steroidogenesis by ACTH was inhibited only by those antibiotics that blocked adrenal protein synthesis—cycloheximide and puromycin. The aminonucleoside of puromycin and 6-dimethylaminopurine, which did not inhibit either protein synthesis or steroidogenesis, caused an even greater fall in hepatic glycogen concentration than puromycin.

These antibiotics failed to stimulate hepatic glycogen depletion in anesthetized animals (*4*); therefore, the effect of inhibitors of protein synthesis on ACTH action under these conditions was studied. Thirty minutes after hypophysectomized rats were anesthetized with pentobarbital, puromycin or cycloheximide was adminstered intraperitoneally. Although hepatic or adrenal glycogen concentration was not diminished, puromycin and cycloheximide inhibited adrenal protein synthesis and still blocked the ACTH stimulation of steroidogenesis (Table 2).

To demonstrate further that the antibiotics affect the ACTH stimulation of steroidogenesis by their action on protein synthesis, the experiment described in Table 3 was performed. When adrenal glands were incubated for 30 minutes to 1 hour in Krebs-Ringer bicarbonate buffer, the concentration of adrenal glycogen fell markedly, in agreement with the observations of Vance *et al.* (*7*). However, the ACTH stimulation of steroidogenesis was not impeded in these glycogen-depleted glands. Furthermore, when

Table 1. Effect of glycogenolysis and inhibition of protein synthesis on the ACTH stimulation of steroidogenesis. Recently hypophysectomized rats were injected intraperitoneally with 10 mg of cycloheximide, 30 mg of aminonucleoside of puromycin, or 14 mg of 6-dimethylaminopurine; 15 minutes later the animals were killed, and glycogen in the liver and adrenals was measured (optical density, per 100 mg, at 620 mμ, as recorded in a Coleman spectrophotometer). Results are means ± standard error (seven animals in each group). In similarly treated rats, ACTH was administered intravenously 15 minutes after treatment with the antibiotics. Corticosterone was assayed from the adrenal-vein blood obtained from 7 to 10 minutes after ACTH treatment (*3*). Simultaneously with the injection of ACTH, that is, 10 min. before the animals were killed, 4μc of C^{14}-labeled algal protein hydrolyzate was injected intravenously; when the animals were killed, the adrenals were removed immediately, and the incorporation of radioactive amino acids into protein was determined (*3*). Results are means ± standard rror determination of four different animals.

Treatment	Glycogen		Cortiscosterone (μg/3 min)	C^{14} incorporated (dpm)*
	Liver (OD/100 mg)	Adrenal (OD/100 mg)		
Adrenal baseline (no ACTH)		1.7 ± .3	0.2 ± .02	4900 ± 300
ACTH + no antibiotic	4.3 ± 0.9	1.7 ± .2†	1.3 ± .2	4978 ± 210
ACTH + puromycin	1.6 ± 0.5	1.8 ± .1	0.2 ± .1	686 ± 52
ACTH + cycloheximide	0.4 ± 0.2	1.7 ± .1	.3 ± .02	200 ± 60
ACTH + aminonucleoside of puromycin	.4 ± .01	1.9 ± .2	1.7 ± .2	6363 ± 697
ACTH + 6-dimethylaminopurine	.2 ± 0.1	1.7 ± .1	1.6 ± .4	4265 ± 124

* Disintegrations per minute per 100 mg of adrenal tissue. † Only six adrenals tested in this group.