tinguer les protéines photo-induites responsables de la caroténogenèse de celles, aussi photo-induites, mais ayant des fonctions autres (photomorphogenèse) que celles associées à la pigmentation[13]. Quant aux différences dans la vitesse de migration entre quelques-unes des protéines de la souche albinos (46-11-68) et celles de la souche jaune (Y 305304), elles sont probablement l'expression de la diversité de leur apparentement génétique[14].

Des expériences immunoélectrophorétiques préliminaires ont été tentées mais, jusqu'à maintenant, il n'a pas été possible de déceler des différences d'arcs entre extraits protéiniques de cultures induites et non-induites. Cela peut refléter soit l'incapacité des enzymes caroténogènes à provoquer une réponse immunologique, soit l'existence d'une différence quantitative, non décelable par les moyens courants, entre la quantité de protéine caroténogène synthétisée constitutivement, à l'obscurité, et celle photo-induite.

La différence entre extraits protéiniques des cultures induites et non-induites est en bon accord avec les résultats des études faisant usage d'inhibiteurs de la synthèse des protéines associée à la caroténogenèse[2,3]. Bien que nos résultats s'accordent avec les études antérieures faisant usage des méthodes génétiques à haute résolution[15] et l'étude par complémentation[16], l'information est encore insuffisante, à l'exception de celle concernant la biosynthèse de la neurosporaxanthine, pour affirmer que la caroténogenèse se poursuit à l'aide du produit d'un seul groupement génique. On peut cependant présumer que cette protéine seule est un aggrégat car au moins 2, voire 4 cistrons, ont été génétiquement associés avec les produits géniques de la région *al*. La prédiction minima est celle de deux activités enzymatiques: les déshydrogénations polyéniques successives et la formation du cycle β-ionone, cette dernière étant, chez d'autres organismes au moins, catalysée enzymatiquement[17].

L'interprétation donnée ci-dessus est aussi en accord avec les analyses biochimiques des pools intermédiaires de caroténoïdes[18–21], indiquant que quelques mutants de la région albinos sont capables de synthétiser une série de caroténoïdes successivement déshydrogénés, à l'exclusion de composés contenant la β-ionone (34508 ou ALS-22-S82) et que d'autres souches peuvent produire la β-ionone mais de petites quantités seulement des caroténoïdes hautement déshydrogénés (*ylo*-b, RES-25-44[8]). Ainsi, une dualité fonctionnelle est suggérée pour le produit des gènes, à savoir la déshydrogénation et la formation du cycle β-ionone[22].

Summary. In an effort to determine the number of proteins responsible for the synthesis of the neutral carotenoids in *N. crassa*, the soluble protein extracts from light-induced and dark-grown cultures of a neurosporaxanthinless strain were compared electrophoretically. The differences relevant to the carotenogenic proteins were identified by reference to an albino polarity mutant. A single, electrophoretically determinable, protein was found to be specific to carotenogenesis. This implication of a single (presumably an enzyme aggregate) protein controlling carotenoid synthesis is discussed and correlated to the existing genetic, complementation and biochemical evidence.

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The Relationship between Urinary Excretion of 17-Hydroxycorticosteroids and Glycosaminoglycans

In 1955 we noticed that the i.v. administration of compound F was followed by an increased urinary excretion of glycosaminoglycans (GAG)[1]. In 1960, Ghata et al.[2] demonstrated in normal individuals a synchronized pattern of excretion of urinary GAG and 17-hydroxycorticosteroids (17-OH-CS). The same authors[2] found that in patients with Addison's disease the urinary 17-OH-CS (but not the GAG) were decreased; both parameters, however, had lost their normal, rhythmic excretion pattern. Moreover[3], they confirmed that administration of ACTH and cortisone produced an immediate increase of urinary GAG. These data were interpreted as indicative of a process of steroid-induced depolymerization of the ground substance, with increased blood levels and urinary excretion of GAG[3].

However, the short interval of time which separates adrenal stimulation (or administration of adrenal steroids) and excretion of GAG cannot be reconciled with a metabolic action on connective tissue. In fact, experimental conditions known to cause degradation of proteoglycans (administration of papain, large doses of vitamin A) require at least 24 h to produce an increase in blood levels and urinary excretion of GAG[4,5].

Recently, we have studied the correlation between urinary excretion of 17-OH-CS and GAG in 2 young, healthy men, members of a 3-man crew participating in 2 simulated Apollo lunar flights.

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Materials and methods. The experiments were performed using a simulator of the Apollo capsule, kept at constant temperature and humidity and at a fixed noise level. The subjects employed performed an initial 14-day control flight to become accustomed to the routine. During the first flight, they performed simultaneously a large number of mechanical and psychological tasks; they ate their meals, performed physical exercises and slept (approximately 8 h/day) at the same time. During the second flight, their activities were completely dissociated; each member had to perform continuous physical and psychological tasks devoting only short and irregularly spaced periods of time to eating and resting. Urine was collected as soon as spontaneously voided by each crew member; the volume and time of emission were noted, and the samples were immediately frozen without addition of preservative. Urinary GAG were measured with a chromatographic method recently described[6] and the 17-OH-CS with the method of GLENN and NELSON[7] after hydrolysis of a urine aliquot with β-glucuronidase. Values were expressed respectively as mg hexuronic acid and mg of 17-OH-CS/volume of each urine sample. Creatinine was measured with the method of FOLIN and WU[8]. Autocorrelation and cross-correlation analysis was used to study the relationship between the 2 variables. To this extent, each day of flight was divided into 6 periods of 4 h each. The weighed average value ($\pm$ S.D.) of excretion of each variable was calculated from the values obtained in the same 4-h period throughout the 14-day flight.

Results and discussion. The concentration of GAG and 17-OH-CS in each urine sample is extremely variable (Figures 1 and 2). However, the excretion of GAG is remarkably constant for the entire period of the experiment, when expressed as mg hexuronic acid/g creatinine and plotted as daily excretion (Figures 1 and 2, inserts).

Table I contains the weighed average values $\pm$ S.D. of GAG and 17-OH-CS excreted by each subject during the various 4-h periods of the 2 flights. The correlation coefficients between the 2 variables for the 2 flights are presented in Table II.

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Table I. Mean $\pm$ S.D. values for urinary 17-hydroxycorticosteroids (17-OH-CS) and glycosaminoglycans (GAG)

Subject			Period					
			6.00–10.00 h	10.00–14.00 h	14.00–18.00 h	18.00–22.00 h	22.00–2.00 h	2.00–6.00 h
Commander	First flight	17-OH-CS	1.91 $\pm$ 0.83	2.31 $\pm$ 1.51	2.72 $\pm$ 1.15	2.03 $\pm$ 1.01	1.54 $\pm$ 0.65	–
		GAG	2.13 $\pm$ 0.81	1.52 $\pm$ 1.17	1.41 $\pm$ 0.53	1.05 $\pm$ 0.30	1.32 $\pm$ 0.24	–
	Second flight	17-OH-CS	0.74 $\pm$ 0.71	2.95 $\pm$ 0.92	2.69 $\pm$ 0.75	2.35 $\pm$ 0.92	1.57 $\pm$ 0.93	1.03 $\pm$ 0.61
		GAG	1.55 $\pm$ 0.07	2.37 $\pm$ 0.78	0.96 $\pm$ 0.49	1.03 $\pm$ 0.47	0.92 $\pm$ 0.58	1.18 $\pm$ 0.35
Navigator	First flight	17-OH-CS	3.21 $\pm$ 1.47	1.68 $\pm$ 0.16	3.27 $\pm$ 1.03	4.68 $\pm$ 1.91	3.77 $\pm$ 3.09	–
		GAG	2.44 $\pm$ 0.86	0.90 $\pm$ 0.07	1.40 $\pm$ 0.25	2.26 $\pm$ 0.88	2.41 $\pm$ 1.77	–
	Second flight	17-OH-CS	3.08 $\pm$ 1.48	3.24 $\pm$ 1.16	3.09 $\pm$ 1.06	3.82 $\pm$ 0.58	2.38 $\pm$ 1.06	3.84 $\pm$ 1.39
		GAG	2.36 $\pm$ 1.00	2.83 $\pm$ 0.06	1.65 $\pm$ 0.55	0.62 $\pm$ 0.46	1.20 $\pm$ 0.34	2.43 $\pm$ 0.80

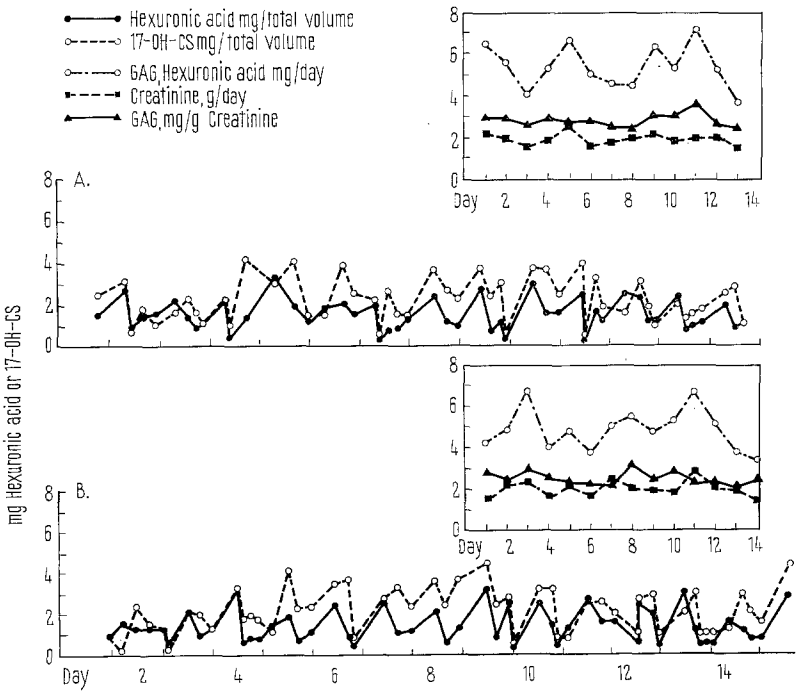

Fig. 1. Concentration of glycosaminoglycans and 17-hydroxycorticosteroids in each urine sample voided spontaneously by the Commander during A. first flight and B. second flight. In the insert the daily excretion of glycosaminoglycans is plotted as mg of hexuronic acid/total volume and as mg of hexuronic acid/g creatinine.

Table II. Correlation coefficients between values of excretion of 17-hydroxycorticosteroids (17-OH-CS) and glycosaminoglycans (GAG)

Subject		Periods					
		6.00–10.00 h	10.00–14.00 h	14.00–18.00 h	18.00–22.00 h	22.00–2.00 h	2.00–6.00 h
Commander	First flight	0.78[b]	0.90[b]	0.65[a]	0.73[a]	0.57[c]	No samples
	Second flight	Small sample	0.66[b]	0.96[b]	0.62[a]	0.69[b]	0.90[a]
Navigator	First flight	0.74[b]	Small sample	Small sample	0.76[a]	Small sample	No samples
	Second flight	0.83[b]	Small sample	0.65[a]	0.24[c]	Small sample	Small sample

[a] Significant at the 0.05 level, $p < 0.05$; [b] Significant at the 0.01 level, $p < 0.01$; [c] Not significant, $p < 0.05$.

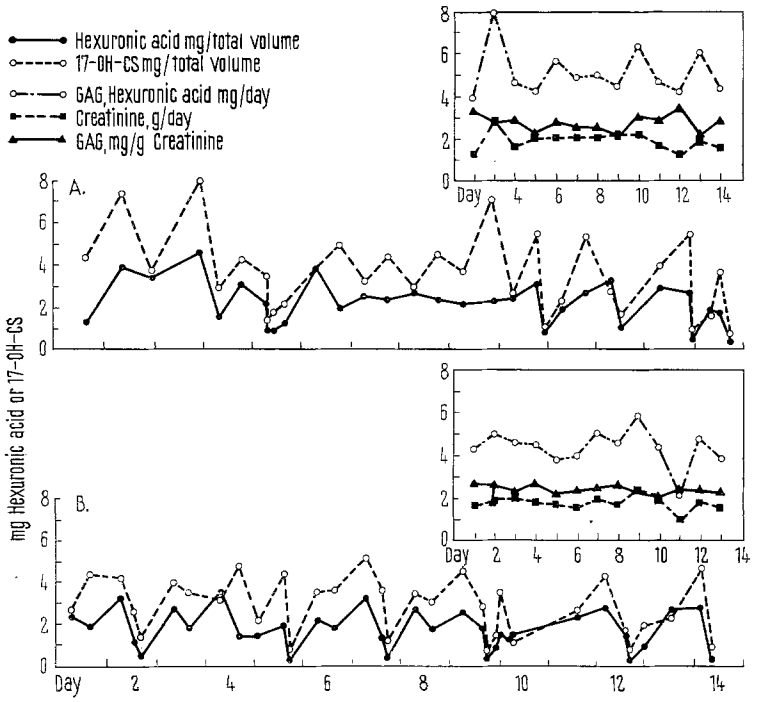

Fig. 2. Same parameters as presented in Figure 1, as applied to the Navigator.

Despite the unusual experimental conditions, the daily excretion of GAG of the 2 subjects during the 2 flights appears to be quantitatively normal and reasonably constant (Figures 1 and 2, inserts). During the first flight, in which the normal circadian rhythm of the subjects should not have been disrupted, the urinary excretion of 17-OH-CS fluctuated more than that of GAG (Table I). This may indicate that the experimental conditions affected the adrenal function more than the metabolism of GAG. Nevertheless, the correlation coefficients indicate a significant relationship between the 2 variables within a given period of time (Table II). During the second flight, the complete randomization of the daily routine did not affect appreciably such a correlation. For the Commander, it remained significant (Table II) and improved during the night period (22.00 h to 2.00 h); for the Navigator, it remained significant (Table II). However, the early morning was the only period of the day that could be adequately studied in this subject.

These results confirm the existance of a significant correlation between urinary excretion of GAG and 17-OH-CS, under normal conditions or conditions in which the circadian rhythm of the individual may be disrupted. However, the data obtained by GHATA et al.[2], the mechanism of action of agents known to mobilize tissue GAG[4,5] and the well documented effects of adrenal hormones on GAG metabolism[9] tend to indicate that the adrenal hormones may play a 'permissive' role in the urinary excretion of circulating GAG[10] rather than a determining role in their mobilization from tissues[11].

Zusammenfassung. Während 2 simulierten, 14 Tage dauernden Raumflügen wurde bei 2 Astronauten untersucht, ob zwischen der Ausscheidung von Glycosaminglycanen und der Ausscheidung der Nebennierenrindensteroide eine Korrelation besteht. Eine signifikante Korrelation zwischen diesen beiden Parametern wurde gefunden.

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