Table 2. Social status of marked Canada geese in relation to migration pattern.

Status	Migration					
	Adult		Yearling		Immature	
	Normal	Reverse	Normal	Reverse	Normal	Reverse
Family*	252	0	1	0	432	1
Gang brood family	11	2			135	0
Paired—no young	60	1	19	0		
Sibling group	5	0	27	3†		
Rejoin parents			34	2‡		
Single	17	1	19	2	23	1
Unknown	132	6	123	6	276	3
Total	477	10	223	13	866	5

*Includes pairs with 1-year-old (or older) young with or without immatures. †Three siblings always together migrated back as a group. ‡Variable behavior—sometimes with parents, sometimes as a single or with a sibling.

ticipated in reverse migration. Yearlings in sibling groups usually stayed together, and one of these groups participated in reverse migration. Yearlings rejoining their parents and new young were variable in behavior and not always in association with their families; these were probably singles when they participated in reverse migration because their parents were not observed with them.

Immatures in identified families did not reverse migrate in proportion to those unclassified as to status or those which were singles ($\chi^2 = 8.34$; 3 d.f.; $P < .02$). Immatures in families were frequently separated temporarily from their parents during feeding because of the large numbers of geese crowded into a small area where food was artificially provided. It was during these feeding periods that most observations of marked birds were made and, as a result, I was indecisive on the status of many young.

Single geese, particularly yearlings, are much more variable in daily roost and feeding locations and flight patterns than are families (*6*). They tend to join in flight with geese taking off near them, whereas adults with young maintain consistent patterns. Return to previously used areas is a major mechanism of reunification of family members. Yearlings are also less traditional in migration patterns than adults (*8*). The strong gregarious nature of geese, coupled with the following behavior of yearling and nonfamily geese, results in some interchange with other flocks and populations and probably accounts for the unusual but regular occurrence of these reverse migrations. Strong attachments to, and seeking of, family members (*6, 9*) probably motivated their return and to the natal marsh or autumn concentration locality.

Of the 28 reverse migrants, 21 returned again in the same autumn to Rochester, Minnesota. Of the other seven, the fate of two was unknown, one was shot in Manitoba and another in Iowa, one was observed in Missouri, and two were observed in Kansas. Irregular migration behavior by yearlings and singles could provide a major mechanism of gene flow, even though one of small proportions, between populations as localized and traditional as Canada geese, which exhibit marked isolationism and show geographic variation in morphology (*4, 10*).

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Mevalonate Metabolism: Role of Kidneys

Abstract. *More than one-half of the amount of mevalonate that is metabolized by pathways not leading to sterols is accounted for by the action of the kidneys. Conversion of mevalonate in vivo to squalene and sterols in the kidneys is confined almost entirely to the proximal and distal convoluted tubules in the cortex. More sterol than squalene is synthesized from mevalonate not only in the liver but also in the kidney.*

We have reported that mevalonate is metabolized in the rat and man not only for the synthesis of polyprenyl substances and sterols, but also on a pathway not leading to sterols (*1, 2*). In order to explain our observations (transfer of C-2 of mevalonate to palmitate and stearate, and the appearance of $^{14}CO_2$ in the breath of animals and man within 1 minute after injection of [5-^{14}C]mevalonate) we proposed a hypothetical metabolic pathway whereby an intermediate derived from mevalonate (possibly 3,3-dimethylallyl pyrophosphate) was diverted from the main sterol-synthesizing reaction sequence via *trans*-3-methylglutaconyl-CoA to ketone bodies and acetyl-CoA in ketogenic organs, the liver, intestine, and kidneys. This metabolic pathway was termed the "*trans*-methylglutaconate shunt" of mevalonate metabolism (*1*).

Table 1. $^{14}CO_2$ in breath of paired control and nephrectomized rats exhaled in 2.5 hours after injection of 2 μmole of *RS*-[5-^{14}C]-mevalonate (MVA).

Experiment	$^{14}CO_2$ (percent of *R*-MVA dose)		$\frac{B}{A} \times 100$
	A*	B†	
1	6.08	2.64	43.4
2	6.31	3.02	47.8
3	6.76	2.58	38.2
4	17.88	5.60	31.4
5	14.35	6.28	43.8

*Sham-operated controls; mean body weight, 284 ± 14 g. †Bilaterally nephrectomized; mean body weight, 286 ± 15 g.

Because of the known avid utilization of mevalonate by the kidneys for the synthesis of squalene and sterols (*2–5*), and because of its known function in ketogenesis (*6*), we have examined by experiments in vivo the possible role of the kidneys in this "shunt" activity and the

renal structures that metabolize mevalonate. The results, reported briefly (*7*), implicate the renal cortex as a very active site of mevalonate metabolism.

Normal, bilaterally nephrectomized and paired sham-operated male Sprague-Dawley rats received intramuscularly immediately after the operation (done under ether anesthesia) 2 μmole (20 μc) of *RS*-[5-^{14}C]mevalonate (Schwarz/Mann) and were put immediately into chambers through which air was being drawn at a rate of 200 ml/min. The air drawn from the chambers was passed through three CO_2 traps, connected in series, and filled, each, with 60 ml of 0.5*N* methylbenzethonium hydroxide (Hyamin) in methanol. The nephrectomized and sham-operated controls were littermates; the experiments on each pair were done on the same day and during the same hours of the day; they were begun at 2 p.m. (*8*). The experiments were terminated 2.5 hours after the injection of mevalonate because previous experiments (*2*) had shown that the utilization of mevalonate per se is complete by that time. We then measured the ^{14}C content of the CO_2 traps in a Packard Tri-Carb scintillation spectrometer (*2*). Blood and organs were analyzed for ^{14}C-labeled unsaponifiable lipids (squalene and sterols) as described (*1, 2, 5, 9*). Sections of kidneys, fixed in formol-saline and embedded in gelatin, were cut in a cryostat at 20 μm thickness and were coated with Kodak AR-10 photographic emulsion for radioautographs which were developed after exposure for 1 day to 3 weeks.

We have reported (*2*) that the amount of $^{14}CO_2$ in the breath of animals and man injected with [5-^{14}C]mevalonate was a measure of the minimum amount of mevalonate metabolized by the "shunt," that is, by a pathway not leading to sterols. We have found now that more than one-half of the "shunt" metabolism of mevalonate is attributable to the action of the kidneys. Although the extent of the "shunt," as judged by the amount of $^{14}CO_2$ found in the breath after injection of [5-^{14}C]mevalonate, varied widely among the animals, nephrectomy always reduced the amount of $^{14}CO_2$ exhaled to less than one-half of that exhaled by the paired sham-operated controls. The mean value of $^{14}CO_2$ exhaled by the nephrectomized rats was only (mean ± standard deviation) 40.9 ± 5.7 percent of that found in the breath of the controls 2.5 hours after the injection of the mevalonate (Table 1). However, between 2.5 and 4 hours after injection of mevalonate, nephrectomized and sham-operated animals exhaled equal amounts of $^{14}CO_2$. We could confirm readily the data of Cuzzopoli *et al.* (*3*) that, in nephrectomized animals, the conversion of mevalonate into unsaponifiable lipids in the liver increased substantially with a concomitant increase in the release of newly synthesized sterols into the blood (Table 2). Our analyses of the unsaponifiable lipids differ substantially from the data of Cuzzopoli *et al.* (*3*) and Hellström *et al.* (*4*) in that we found that even in the kidneys [^{14}C]squalene was a minor component of the unsaponifiable lipids after injection of [5-^{14}C]-mevalonate (Table 2). The investigators quoted (*3, 4*) have reported that the major labeled constituent in the unsaponifiable lipids of the kidneys, 0.5 to 2 hours after injection of [2-^{14}C]mevalonate, was squalene. It is uncertain whether differences in strains of animals or in experimental techniques are responsible for the differences between our observations and those of Cuzzopoli *et al.* (*3*) and Hellström *et al.* (*4*).

Table 2. Effect of nephrectomy on conversion of [5-^{14}C]mevalonate into unsaponifiable lipids in rats. The data are from the same animals, 2.5 hours after injection of mevalonate, as shown in Table 1. The results are mean values ± standard deviation; dpm, disintegrations per minute.

Fraction	Kidneys* (10^3 dpm/g)	Liver* (10^3 dpm/g)	Blood (10^3 dpm/ml)
	Controls		
Unsaponifiable lipids	4111 ± 120	132 ± 1.8	25 ± 3
Squalene†	168 ± 14	5.2 ± 1.7	0.05 ± 0.01
	Nephrectomized		
Unsaponifiable lipids		730 ± 25	96 ± 17
Squalene†		60 ± 13	0.78 ± 0.24

*Mean weight of the kidneys was 2.43 ± 0.13 g and of the liver in control and experimental animals was identical at 11.2 ± 1.0 g. †The remainder of the unsaponifiables were accounted for by sterols; more than 80 percent of the sterols in all specimens was cholesterol.

Radioautographs of the kidneys of animals injected with [5-^{14}C]mevalonate showed that the water-insoluble ^{14}C was almost entirely confined to the cortex; radioactivity in the medulla was just detectable after 3 weeks' exposure (Fig. 1A). In the cortex, the radioactivity was found predominantly in the proximal and distal convoluted tubules, relatively little being present in the glomeruli (Fig. 1B). About 90 percent of the radioactivity in the kidney sections was extractable by immersion of the sections in a mixture of chloroform and methanol (2 : 1, by volume) at room temperature. These observations differ from those of Raskin and Siperstein (*10*), who reported, from a comparison in vitro of isolated glomeruli and renal cortical tubules, that 95 percent of the utilization of mevalonate for synthesis of unsaponifiable lipids by the renal cortex was attributable to the activity of glomeruli. If Raskin and Siperstein's observations (*10*) were valid for in vivo conditions, one would have expected the glomeruli to stand out as black clusters in the radioautographs. The difference between our observations and those of Raskin and Siperstein (*10*) may be attributed to a difference in the route by which mevalonate reaches the renal tubules in vivo and in vitro. In vivo, mevalonate must be conveyed to the tubules by the glomerular filtrate and reabsorbed from there through the inner

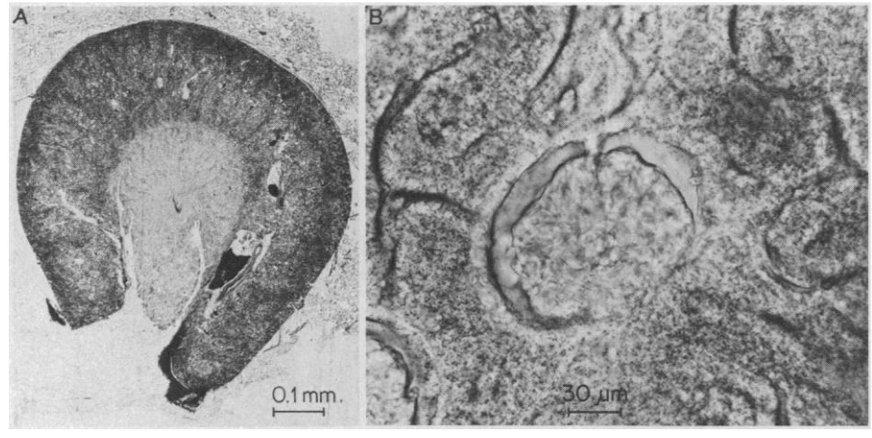

Fig. 1. Radioautographs of unstained kidney section from a normal rat (260 g) 2.5 hours after injection of 20 μc of *RS*-[5-^{14}C]mevalonate. (A) Cross section of kidney. The radioactivity is confined almost entirely to the cortex. (B) High-power view of cortex from the same section as in (A). The glomerulus in the center has few reduced silver grains over it; the remaining structures are mostly proximal convoluted tubules that contained most of the radioactivity.

plasma membrane, whereas in experiments in vitro with isolated tubules primarily the outer tubular membranes are exposed to mevalonate.

The fact that *S*-mevalonate is excreted in the urine (*11*), whereas *R*-mevalonate is not, except after very large doses, such as after 2.7 μmole per gram of body weight in the rat (*1*), suggests the existence of a saturable and stereospecific transport system into the renal tubules for *R*-mevalonate.

Mevalonate is known to be circulating in the blood although at a low level (*12*). Our observations suggest that an impairment of the renal "clearance" of blood mevalonate by either of two metabolic pathways (synthetic and "shunt") could account for the hypercholesterolemia associated with some diseases of the kidneys, since such impairment might lead to increased hepatic synthesis and increased release of cholesterol into the blood. Edgren and Hellström (*12*) reported that, in aminoglycoside-induced nephrosis in rats, associated with hypercholesterolemia, there was a much increased utilization of mevalonate by the liver and an increase in the release of newly synthesized cholesterol into the blood, similar to that seen after nephrectomy.

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Paracytic (Syndetocheilic) Stomata in Carboniferous Seed Ferns

Abstract. *Stomata of the paracytic type have been discovered on the lower pinnule surface of the Carboniferous seed fern foliage species* Alethopteris sullivanti. *The stomatal complex is not visible when the cuticle is in place. This is apparently the first report of paracytic stomata in Carboniferous plants.*

Stomatal types are of interest to taxonomists and structural botanists because of their taxonomic usefulness and, in some instances, because of their phylogenetic significance (*1*). Paleobotanists have generally followed the terminology established by Florin (*2*), who recognized two fundamental stomatal types—haplocheilic and syndetocheilic. Since these terms incorporate developmental patterns in their definitions, their usage when applied to fossils necessarily involves one or more inferences (*3*). We have used the purely descriptive term, paracytic, followed in parentheses by the inferential one, syndetocheilic. The latter term is used because of its familiarity to paleobotanists; in this instance we consider the terms paramesogenous (*3*) and mesoparacytic (*4*) equivalent to syndetocheilic.

To the best of our knowledge, this is the first report of paracytic (syndetocheilic) stomata in a Paleozoic seed fern. The stomata were discovered on the lower (abaxial) surface of the distinctive foliage species *Alethopteris sullivanti*, preserved in middle Pennsylvanian coal balls collected near Cayuga, Indiana. Guard cells average 27 μm in length and are completely enclosed by two subsidiary cells (Fig. 1a). The common end walls of the subsidiary cells meet at the midline through the long axis of the stoma and at each pole. The outer walls of the guard cells are thickened and, consequently, darker in color. The presumed cytoplasmic contents of the guard cells are sometimes withdrawn from the outer wall, leaving a clear area adjacent to the thickened outer wall (Fig. 1c, unlabeled arrow).

Alethopteris sullivanti is one of the most completely known pteridosperm foliage species, and has been studied by Leisman (*5*) and by Faulwell and Schabilion (*6*). In neither study, however, were paracytic stomata detected. We attribute this to a lack of paradermal sections, which would pass just below the surface and which would reveal the slightly sunken guard and subsidiary cells. In both of the previous reports (*5*, *6*), stomata were studied primarily from cuticles macerat-

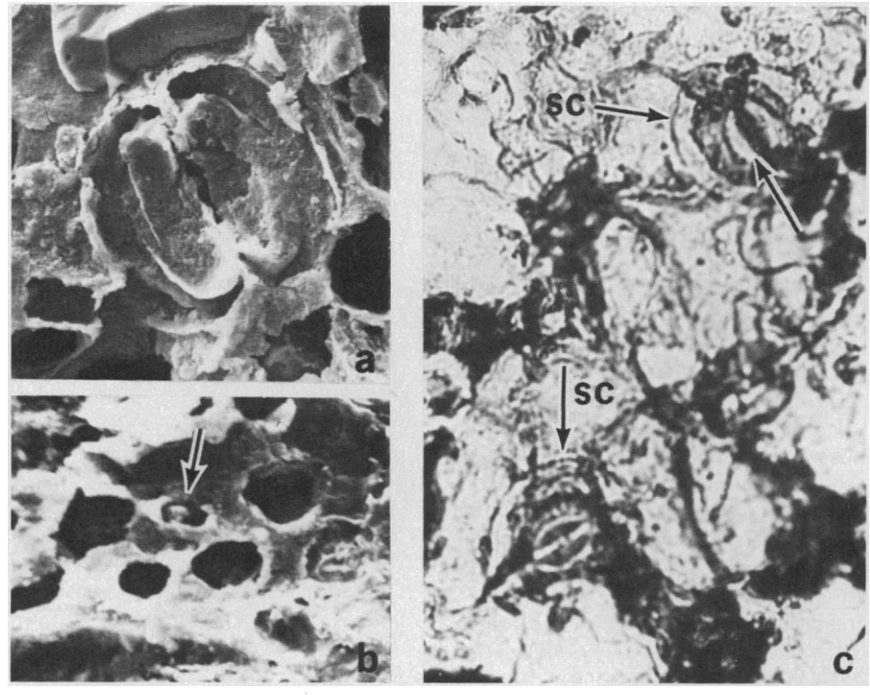

Fig. 1. (a) Stomatal complex of *A. sullivanti* showing subsidiary cells completely enclosing guard cells (× 850). (b) Pinnule surface showing ring of papillae bases surrounding stomatal aperture (arrow). Stomatal complex is out of sight below surface (× 550). (c) Light microscope photograph of lower epidermis beneath cuticle; *SC* arrows touch subsidiary cells; clear area (unlabeled arrow) was created by withdrawal of guard cell cytoplasm from thickened outer guard cell wall; stoma are closed (× 480).