formed essentialy as described [M. E. Greenberg and E. B. Ziff, *Nature (London)* **311**, 433 (1984)], but with modifications described elsewhere (*19*). Each nitrocellulose filter "slot" contained 1.0 μg of denatured, immobilized DNA. After hybridization with a solution containing 10^7 count/min per milliliter of RNA sample, filters were washed and exposed to x-ray film for 13 days at −70°C with intensifying screen, and results quantitated by densitometry. The observation of a considerably more intense signal with a ribosomal RNA probe than with probes for mRNA transcripts (Fig. 3) demonstrates that for the latter, immobilized DNA was in excess.

15. The 5′-deletion mutants of pPRL-CAT and pGH-CAT were constructed as follows. To generate pPRL-CAT deletions containing 958 and 618 nucleotides, respectively, of prolactin 5′-flanking sequences, pPRL-CAT was digested with Sma I and then Sph I or Sst I, then treated to make the ends blunt and religated. Further 5′ deletions were prepared by Bal 31 digestion initiated in the polylinker of the −618 deletion. All deletions share the same vector sequences 5′ to the prolactin deletion end point. Deletion end points were determined precisely by dideoxy sequencing [E. Y. Chem and P. J. Seeburg, *DNA* **4**, 165 (1985)]. Plasmid pΔGH-CAT, with 0.236 kb of growth hormone–gene 5′-flanking sequence, contains the pGH-CAT Bgl II–Bam HI fragment cloned into the pUC 9 Bam HI site.
16. CAT activity was assayed essentially as described [C. M. Gorman, L. F. Moffat, B. H. Howard, *Mol. Cell. Biol.* **2**, 1044 (1982)], except that extracts were incubated at 65°C for 5 minutes to inactivate an endogenous deacetylase [M. Mercola, J. Goverman, C. Mirell, K. Calame, *Science* **227**, 266 (1985)]. Incubation was at 37°C for 8 to 12 hours, in the linear range of the assay. Increased levels of CAT enzymatic activity were detectable as early as 12 hours after cell fusion.
17. The following results imply that activation also does not require the 38 bp of prolactin gene body sequences present in these pPRL-CAT derivatives. Plasmid pPΔmCAT contains the prolactin DNA sequences between Pst I sites at −1957 and −11, linked through a Hind III linker at −11 to the Hind III site of the Hind III–Bam HI CAT gene fragment, cloned into pUC 9. In fused heterocultures, equal levels (*11*) of CAT mRNA were yielded by pPΔmCAT and pPRL-CAT.
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32. We thank the following colleagues for supply of materials: pRSV-CAT, B. Howard; mouse pituitary RNA and mouse prolactin and growth hormone cDNA clones, D. I. H. Linzer and F. Talamantes; and pXC-1, R. V. Guntaka. This work was supported by NIH grant GM36847.

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Three Recessive Loci Required for Insulin-Dependent Diabetes in Nonobese Diabetic Mice

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A polygenic basis for susceptibility to insulin-dependent diabetes in nonobese diabetic (NOD) mice has been established by outcross to a related inbred strain, nonobese normal (NON). Analysis of first and second backcross progeny has shown that at least three recessive genes are required for development of overt diabetes. One, *Idd-1*^{s}, is tightly linked to the *H-2K* locus on chromosome 17; another, *Idd-2*^{s}, is localized proximal to the *Thy-1/Alp-1* cluster on chromosome 9. Segregation of a third, *Idd-3*^{s}, could be shown in a second backcross. Neither *Idd-1*^{s} nor *Idd-2*^{s} could individually be identified as the locus controlling insulitis; leukocytic infiltrates in pancreas were common in most asymptomatic BC1 mice. Both F1 and BC1 mice exhibited the unusually high percentage of splenic T lymphocytes characteristic of NOD, suggesting dominant inheritance of this trait. The polygenic control of diabetogenesis in NOD mice, in which a recessive gene linked to the major histocompatibility complex is but one of several controlling loci, suggests that similar polygenic interactions underlie this type of diabetes in humans.

NONOBESE DIABETIC (NOD) MICE derived by Makino *et al.* from the non-inbred ICR strain are a model for insulin-dependent diabetes (Idd) in man (*1*). Both cellular and humoral autoimmunity against pancreatic β cells appears to be a central feature in pathogenesis (*2, 3*). Insulitis, a leukocytic infiltration of the pancreatic islets, is a salient histopathological lesion (*1, 4*). Insulitis was found at approximately 40% frequency in reciprocal [(NOD × NON)F1 × NOD] backcross mice at 9 weeks of age, suggesting control by a single recessive trait inherited from NOD mice (*4*). Subsequently, a diabetogenic recessive gene was associated with the unique NOD *H-2* haplotype on chromosome 17 (Chr 17) when an outcross/backcross analysis was performed between NOD and C3H mice (*5*). However, the low frequency of overt diabetes obtained in this analysis suggested the involvement of more than one NOD-derived recessive gene.

Breeding stock of the NOD strain and a related diabetes-resistant strain, nonobese normal (NON) were used at inbred generation F32 and F35, respectively. We have continued inbreeding and designate our sublines as NOD/Lt and NON/Lt; these inbred strains differ at numerous loci including the major histocompatibility complex (MHC), *H-2* (*4–6*). Consistent with the previously observed diabetes incidence of 80% in females and 10% in males (*1*), NOD/Lt females exhibit a diabetes incidence of >90% by 10 months of age; however, NOD/Lt males exhibit a higher than reported diabetes incidence, averaging 50 to 70% by 10 months when fed diet formulation 96W (Emory Morse Co., Guilford, Connecticut). F1 mice (23 males and 24 females) from reciprocal NOD/Lt × NON/Lt outcrosses were studied for development of hyperglycemia over a 12-month period. In contrast to the high incidence of hyperglycemia in parental NOD/Lt mice of both sexes by 12 months of age, F1 mice of both sexes were uniformly diabetes-resistant. NON/Lt can be distinguished from NOD/Lt mice by the number and functions of T lymphocytes in peripheral blood and spleen, as well as by their *Thy-1* phenotypes [Thy-1.1 and -1.2, respectively (6)]. NON/Lt mice develop T-lymphocytopenia associated with an age-dependent decline in responsiveness to concanavalin A whereas NOD mice show a persistent T cell hyperplasia associated with strong T cell mitogenic responses (*7*). However, the numbers and mitogen responsiveness of T lymphocytes in all F1 mice examined were NOD-like (*7*). Indeed, although pancreatic islet structure was intact and numbers of granulated β cells were normal, focal pancreatic lymphocytic infiltrates in perivascular and periductular areas, often abutting islets at one pole (but quite distinct from the insulitis observed in NOD parental mice), were noted in five of nine F1 males and nine of ten females examined histologically at 12 months. Thus, both recessive and dominant traits inherited from NOD were associated with pathogenesis.

In the present study, the genetic polymorphisms distinguishing NOD/Lt from

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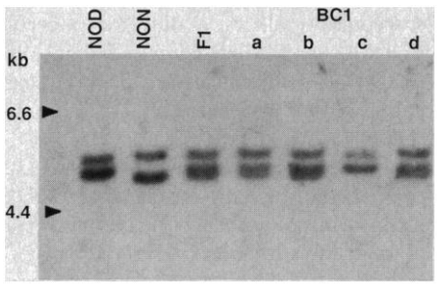

Fig. 1. Southern blot of genomic, Pvu II–digested liver DNA showing the polymorphism distinguishing NOD, NON, and F1 mice at the *P450-3* locus on Chr 9. Two bands are observed; the upper 5.0-kb fragment is common to both strains whereas the anodal lower band is polymorphic (4.8-kb in NOD and 4.6-kb in NON). Lanes a to d show representative RFLP patterns observed in diabetic BC1 mice with the following genotypes: lane a, *Thy-1*^{b}/*Alp-1*^{b}/*Mod-1*^{ab}; lane b, *Thy-1*^{ab}/*Alp-1*^{ab}/*Mod-1*^{ab}; lane c, *Thy-1*^{b}/*Alp-1*^{b}/*Mod-1*^{b}; and lane d, *Thy-1*^{ab}/*Alp-1*^{ab}/*Mod-1*^{b}. These segregation data collectively indicate that *Idd-2* must be located proximal to the *Thy-1*/*Alp-1* markers.

NON/Lt mice on Chr 1, 3, 5, 9, 15, and 17 were utilized to determine the number and the chromosomal localization of the NOD recessive diabetogenic genes. In addition to the reported polymorphic differences between the NOD/Lt and NON/Lt (*6*), we found a restriction fragment length polymorphism (RFLP) at the *P450-3* locus on Chr 9 with Pvu II–digested genomic DNA probed with the full-length complementary DNA clone pP$_3$450FL (*8*). The polymorphic Pvu II fragment in NOD mice is 4.8 kb and in NON mice is 4.6 kb (Fig. 1).

A total of 200 first backcross (BC1) mice were produced from reciprocal backcrosses of F1 mice to the diabetes-susceptible NOD/Lt parental strain. Three mice died of causes other than diabetes and three more died between 8 and 10 months of undiagnosed causes. Only 19 of the BC1 mice (6 males, 13 females) developed overt diabetes (glycemic changes) during the 12-month observation period. This diabetes incidence (9.5%) contrasts sharply with an expected 50% diabetes incidence (uncorrected for penetrance) if a single recessive MHC-linked gene controlling insulitis were segregating and is consistent with a ratio expected for at least three unlinked autosomal recessive genes. All 19 diabetic mice were homozygous for the *H-2K*^{d} allele of NOD; 18 were *I-E*^{o} like NOD (Table 1). The one exception, a *K*^{d}*I-E*^{k} recombinant female, was the first BC1 diabetic to be detected. The classification of this female by MHC-typing of peripheral blood and splenic leukocytes was independently confirmed by RFLP analysis of Bam HI–digested genomic DNA hybridized with an E$_\beta$ specific probe (*9*). The finding that diabetogenesis was dependent upon homozygosity for the NOD *H-2K*^{d} marker allele confirmed the observation of Hattori *et al.* (*5*) that a diabetogenic recessive trait is MHC-linked. Our data establish that this locus [provisionally named *Idd-1*, in accordance with the rules recommended by The Committee on Standardized Genetic Nomenclature for Mice (*10*)] is tightly linked to the *H-2K* end of the MHC. NOD mice possess the susceptibility allele, *Idd-1*^{s}, and NON a resistance allele, *Idd-1*^{r}. Our evidence does not allow determination of whether the diabetogenic recessive gene is within the MHC, or between the centromere and the *K* end of the *H-2* complex. Since the first mouse to develop diabetes was a recombinant between *H-2K* and *I-E*, the diabetogenic recessive locus would have to be between these two markers if it were within the MHC. The likely position for this recessive gene would be the unique *I-A*$_\beta$ region of NOD (*11*). However, *I-A* genes in heterozygotes are expressed codominantly. Such codominant expression might in part explain why all F1 and BC1 mice analyzed in the present study had the high percentages of splenic T cells characteristic of the NOD strain (Table 2).

Screening the BC1 mice for other polymorphic genetic markers distinguishing NOD/Lt from NON/Lt at Chr 1 *(Idh-1)*, Chr 3 *(Car-2, Amy-1)*, Chr 5 *(Dao-1)*, Chr 9 *(Thy-1, Alp-1, P450-3, Mod-1)*, and Chr 15 *(Gpt-1)* indicated a second recessive diabetogenic gene on the proximal end of Chr 9 located within the 25-centiMorgan (cM) segment between the centromere and *Alp-1* (Table 1). This locus and its respective alleles are provisionally designated *Idd-2*^{s} in the NOD strain, and *Idd-2*^{r} in the NON strain. Thirteen of the diabetic mice shown in Table 1 (including two heterozygotes for *Alp-1*) were also typed for *Thy-1* located 1 cM proximal to *Alp-1*. Since no recombination was observed between these two tightly linked markers (all mice with the NOD b allele at *Alp-1* had the NOD *Thy-1*^{b} allele as well), the *Idd-2* locus is tentatively positioned proximal to the *Thy-1*.

The NON/Lt strain could be differentiated from the NOD/Lt strain in regard to the complete absence of insulitis in the former strain, even after 2 years of age. In NON/Lt mice at 1 year of age and older, focal mononuclear cell infiltrates were observed in association with ducts, blood vessels, and acini, and occasionally, at one pole of an islet. However, insulitis (mononuclear cell infiltration into the islet capsule, with associated islet structural erosion), was not observed in this strain. Histopathologic analysis of the pancreases of nondiabetic BC1 mice surviving to 1 year of age showed a high frequency of leukocytic infiltrates that were completely independent of whether the mice typed homozygous or heterozygous for NOD markers for either or both the *Idd-1*^{s} and *Idd-2*^{s} alleles. Sections of Bouin's-fixed, paraffin-embedded pancreases from each mouse, sampled at three different levels 1000 μm apart, were screened for lymphocytic infiltration; staining with aldehyde fuchsin to detect granulated β cells allowed determination of β cell cytopathology associated with insulitis. Approximately 25 to 35 islets per mouse were assessed. Pancreases of only 17% of asymptomatic BC1 mice were free of islet-associated lesions and these included all possible (inferred) *Idd* genotypes (Table 3). Focal leukocytic infiltrates were observed in 83% of the asymp-

Table 1. Genetic profile of diabetic BC1 mice showing localization of two diabetogenic recessive genes on NOD Chr 9 and 17. Phenotypes of apolipoprotein-1 (*Alp-1*) and supernatant malic enzyme (*Mod-1*) were determined on Titan III cellulose acetate plates (Helena Laboratories, Inc.) as described (*13*). Allele a designates a fast anodal electrophoretic variant, allele b a slow variant of the respective protein. RFLP alleles at *P450-3* were determined by Southern blot analysis of 10 μg of liver DNA digested with Pvu II and probed with nick-translated pP$_3$450FL plasmid as described (*8*). D, 4.8-kb fragment in NOD; N, 4.6-kb fragment in NON; DN, both fragments in F1. The MHC haplotype of NOD is *K*^{d} *I-A*$^{?}$ *I-E*^{o} *Ss*^{h} *Slp*^{o} *D*^{b} and of NON is *K*^{b} *I-A*^{o} *I-E*^{k} *Ss*^{h}*Slp*^{o} *D*^{b}. The *I-A* of NOD is unique (*11*) and a haplotype designation is pending; the *I-A* of NON is as yet uncharacterized. *H-2K* and *I-E* were typed in a microcytotoxicity assay (*14*) by means of monoclonal antibodies against *K*^{d} (31-3-4), *K*^{b} (28-13-3S), and *E*^{k} (17-3-3) determinants. All BC1 mice were typed; this table shows the genotypes of the 19 mice that developed diabetes.

Strain	Chromosome 9			Chromosome 17		Number observed
	Alp-1	*P450-3*	*Mod-1*	*H-2K*	*I-E*	
NOD	b	D	b	d	o	
NON	a	N	a	b	k	
F1	ab	DN	ab	db	k	
BC1	b	D	b	d	o	11
	b	D	ab	d	o	1
	b	DN	ab	d	o	3
	ab	DN	b	d	o	2
	ab	DN	ab	d	o	1
	b	D	ab	d	k	1

Table 2. Inheritance of T-lymphocyte hyperplasia.

Strain	Phenotype		Percent viable splenic leukocytes (*n*)*		Age range (weeks)
	H-2K	*Thy-1*	T cells	B cells	
NOD	dd	.2/.2	42.6 ± 1.95 (5)	53.2 ± 1.12 (5)	30
NON	bb	.1/.1	15.1 ± 2.52 (8)	57.2 ± 2.51 (8)	20
F1	bd	.1/.2	39.5 ± 2.37 (4)	46.6 ± 2.27 (4)	26
BC1	dd	.2/.2	37.9 ± 1.99 (31)	42.4 ± 1.84 (31)	14–44
BC1	dd	.1/.2	33.3 ± 1.56 (7)	48.2 ± 2.67 (7)	12–42
BC1	db	.2/.2	35.4 ± 2.21 (13)	50.8 ± 2.39 (13)	36–44
BC1	db	.1/.2	29.0 ± 2.16 (11)	52.1 ± 2.96 (11)	36–44

*Values represent the mean percentage ± SEM of viable splenic leukocytes for the indicated number of mice (*n*). Separate aliquots of 2×10^6 splenocytes were incubated with or without mouse monoclonal antibody to Thy-1.1 (anti–Thy-1.1) or anti–Thy-1.2 for 30 minutes, washed, and then stained for an additional 30 minutes with fluoresceinated goat anti-mouse immunoglobulin, prior to enumeration in an Ortho cytofluorograph. T lymphocyte levels were calculated by subtracting the percentage of splenocytes staining positive for surface immunoglobulin in the absence of anti–Thy-1 from those incubated with anti–Thy-1 followed by anti-mouse immunoglobulin. T cell percentages of F1 and all BC1 genotypes were significantly different from NON ($P \leq 0.001$), but F1 percentages were not significantly different from NOD. Both groups of BC1 mice heterozygous at *Thy-1* had a significantly lower percentage in comparison to NOD ($P \leq 0.003$).

tomatic BC1 mice of both sexes. Although primarily concentrated peripherally to structurally intact islets (periductular and perivascular as observed in F1 mice), lymphocytic penetration into variable numbers of islets (insulitis) was clearly observed in 27% of the pancreases of asymptomatic mice, again without correlation to (inferred) homozygosity for either or both of the *Idd-1*^{s} and *Idd-2*^{s} alleles. Although insulitis was scored in only a few of the islets most of the islets with or without the periinsular leukocytic aggregations were structurally intact and filled with well-granulated β cells, thus reflecting the normoglycemic status of the mice.

If only two diabetogenic recessive genes inherited from the NOD strain are required for diabetogenesis, then a maximum of 25% of BC1 mice should have developed diabetes (assuming full penetrance). On the contrary, if three recessive *Idd* alleles were required, a theoretical maximum incidence of 12.5% would be expected at BC1 (very close to the 9.5% observed). We tested the hypothesis that the 27% (37/139) of nondiabetic BC1 mice that were homozygous for NOD-type marker alleles on Chr 9 and 17 (and therefore presumably also for *Idd-1*^{s} and *Idd-2*^{s}) remained diabetes-free due to heterozygosity at a third recessive locus *(Idd-3)*. Nondiabetic BC1 males homozygous for NOD Chr 9 and 17 markers were selected for a second backcross (BC2) to NOD females. If these selected BC1 breeders were diabetes-resistant because of heterozygosity at a third locus (*Idd-3*^{s}/*Idd-3*^{r}), then the maximum incidence of overt diabetes in the BC2 generation would theoretically be 50% (assuming complete penetrance). A diabetes incidence of 46% (27 of 59 mice, 12 males and 15 females) was obtained by 10 months of age; this strongly supported the conclusion that NOD mice differ from NON mice by susceptibility alleles at only the one additional locus. No linkage of *Idd-3*^{s} to informative biochemical genetic markers segregating in BC2 at Chr 1, 3, 5, or 15 could be shown.

Our results indicate that the genetic basis of diabetes susceptibility in the NOD/Lt strain versus resistance in the NON/Lt strain is attributable to polygenic interactions of at least three NOD recessive genes and possibly also of (co)dominant determinants inherited from the NOD/Lt strain that control T cell hyperplasia. Makino *et al.* (*4*) screened pancreases of 9-week-old prediabetic (NON × NOD)F2 and BC1 mice for insulitis; their data suggested that genetic control of diabetogenesis in these closely related strains was mediated by a single autosomal locus producing presence or absence of insulitis. Given the widespread distribution of leukocytic infiltrates in pancreases of 1-year-old (NON × NOD)F1 and BC1 mice found in the present study, it seems unlikely that *Idd-1*^{s}, *Idd-2*^{s}, or *Idd-3*^{s} individually represents the recessive locus controlling the presence of insulitis. The pancreatic aggregations may reflect the dominant T cell lymphoproliferative drive inherited from NOD. In F1 and nondiabetic BC1 individuals, these aggregates were predominantly localized at the islet capsule perimeters rather than infiltrating into the core of the islets (insulitis).

Our study indicates that a complex polygenic interaction is required for eliciting sufficient β-cell destruction to be reflected by widespread insulitis and development of overt diabetes. This study has only elucidated diabetogenic genes that distinguish NOD/Lt from NON/Lt mice. That these closely related strains share numerous diabetogenic susceptibility loci was clearly illustrated when outcross/backcross analysis was performed between NOD and an unrelated strain, C57BL/KsJ, selected because of its well-established sensitivity to diabetogenic chemicals and obesity genes (*7*). Only one BC1 female of 115 mice (0.9%) produced by backcross of (C57BL/KsJ × NOD/Lt)F1 to NOD/Lt mice developed diabetes, indicating that NOD/Lt and C57BL/KsJ mice differ at a minimum of six diabetogenic recessive loci.

This polygenic basis of insulin-dependent diabetes inheritance in mice is similar to that in rats (*12*) and clearly indicates that analysis of a susceptibility genotype for diabetes mellitus in humans should not be limited only to analysis of HLA-linked genes. Location of non-MHC–linked diabetogenic genes in NOD mice should prove valuable in suggesting other human chromosomes that may carry loci affecting autoimmunity against pancreatic β cells. In humans, the

Table 3. Dissociation of pancreatic leukocytic infiltrates from *Idd-1*^{s} and *Idd-2*^{s} diabetogenic alleles inherited from NOD. Data are from 139 aglycosuric and normoglycemic BC1 mice surviving to 1 year of age. Phenotypes for *Alp-1* and *Mod-1* markers are described in Table 1.

Marker genes			Linked diabetogenic genes*		Number of mice with degree of leukocytic infiltration†				Total
Alp-1	*Mod-1*	*H-2K*	*Idd-1*	*Idd-2*	0	1	2	1 and 2	
b	b	d	s	s	2	5	22	8	37
b	ab	d	s	s	1	1	3	1	6
ab	ab	db	sr	sr	3	2	14	1	20
ab	b	db	sr	sr	0	0	5	0	5
b	b	db	sr	s	11	0	16	8	35
ab	ab	d	s	sr	5	2	11	3	21
ab	b	d	s	sr	0	2	2	1	5
b	ab	db	sr	s	1	1	6	2	10
				Total	23	13	79	24	139

*s = NOD susceptibility allele; r = NON resistance allele. Since none of these mice were diabetic, the genotypes at *Idd-1* and *Idd-2* are inferred on the basis of marker genes. †Intensity of leukocytic infiltrates are scored as follows: 0 = none, with no visible lesions to islets; 1 = insulitis (penetration of lymphocytes into the islet capsule, with islet cells exhibiting cytoarchitectural disarray and erosion of beta cell mass); 2 = periductular and perivascular lymphocytic aggregations, often localized at one pole or surrounding islets which are completely normal in structure and aldehyde fuchsin staining properties.

THY-1 and *ALP-1* loci are found on the long arm of Chr 11; it is therefore reasonable to suggest this chromosome as a potential site for a diabetogenic locus analogous to *Idd-2*.

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Modern Turtle Origins: The Oldest Known Cryptodire

EUGENE S. GAFFNEY, J. HOWARD HUTCHISON, FARISH A. JENKINS, JR., LORRAINE J. MEEKER

The discovery of a turtle in the Early Jurassic (185 million years before present) Kayenta Formation of northeastern Arizona provides significant evidence about the origin of modern turtles. This new taxon possesses many of the primitive features expected in the hypothetical common ancestor of pleurodires and cryptodires, the two groups of modern turtles. It is identified as the oldest known cryptodire because of the presence of a distinctive cryptodiran jaw mechanism consisting of a trochlea over the otic chamber that redirects the line of action of the adductor muscle. Aquatic habits appear to have developed very early in turtle evolution. *Kayentachelys* extends the known record of cryptodires back at least 45 million years and documents a very early stage in the evolution of modern turtles.

THE EVOLUTIONARY HISTORY OF turtles extends at least to Late Triassic time (200 million years before present), but the fossil record of their early diversification is incomplete. A substantial structural and temporal hiatus exists between the most primitive known form, *Proganochelys quenstedti* of the Late Triassic (middle Keuper of Germany) (*1*), and turtles with essentially modern features, which first appeared in the Late Jurassic (140 million years before present) (*2*). *Proganochelys* has a shell and is hypothesized as the sister group of all other turtles (*3*), but the skull has relatively few chelonian features. There are no characters of *Proganochelys* that indicate that this genus belongs to either the cryptodires or the pleurodires, the two groups of modern turtles. Both cryptodires and pleurodires possess distinctive specializations of the skull and postcranial skeleton consistent with the interpretation that they are monophyletic, and together they are united in a single monophyletic taxon, the Casichelydia (*4, 5*). The discovery of an Early Jurassic (185 million years before present) cryptodire (Fig. 1) extends the known history of the cryptodires back more than 45 million years and documents an intermediate stage in the evolution of modern turtles; the skull and shell structure of this new form represents an appropriate ancestral morphotype for all Cryptodira.

The systematics:

Order Testudines
Gigaorder Casichelydia
Megaorder Cryptodira
Family Kayentachelyidae, new
Kayentachelys, new genus

Type species: *Kayentachelys aprix*, new species.

Known distribution: Early Jurassic, Arizona, United States.

Etymology: Kayenta, for the Kayenta Formation; *chelys*, Greek for turtle.

Diagnosis: As for species.

Kayentachelys aprix, new species

Type specimen: Museum of Northern Arizona V1558.

Locality: Gold Spring, Adeii Eechii Cliffs, Coconino County, Arizona (35°45′35″N, 111°04′51″W).

Horizon: Silty facies of the Kayenta Formation, Early Jurassic.

Etymology: *aprix*, Greek for tight, in reference to the fused basicranial articulation.

Referred specimens: MNA V1559-V1570; V2664. MCZ 8914-8917.

Diagnosis: A combination of primitive and advanced characters (*6*). Primitive amniote characters: pterygoid teeth, interpterygoid vacuity; prootic exposed ventrally. Primitive chelonian characters: nine costals; epiplastron with dorsal process. Derived casichelydian characters: antrum postoticum; fused basipterygoid articulation; 11 peripheral bones. Derived cryptodiran characters: processus trochlearis oticum; processes pterygoideus externus projecting posteriorly with a flat, vertical plate (*7*).

Turtles have often been cited as examples of "living fossils," a group that is structurally conservative throughout its history. In fact, however, this viewpoint is erroneous. Although all turtles appear superficially similar because they have a shell, there have been fundamental cranial and postcranial changes during their history. Cryptodires and pleurodires (Fig. 2), the two groups of living turtles, have independently evolved different trochlear mechanisms that redirect the main tendon of the jaw adductor muscle (*8*). As a result, the adductor, which has expanded posteriorly relative to the jaw joint, maintains a vertical line of action during jaw closure. In cryptodires the trochlea is formed by a thickened protuberance on the anterodorsal portion of the prootic and quadrate, whereas in pleurodires the trochlea is a lateral process of the pterygoid. *Kayentachelys* has the cryptodire trochlear condition. Another characteristic of cryptodires is an extensive fusion of the palatoquadrate and neurocranium that encloses the middle ear. Primitive amniotes and *Proganochelys* lack these advanced characters of the ear region and also have an open basipterygoid articulation. *Kayentachelys* represents an intermediate stage in that it has a fused basipterygoid articulation but lacks the distinctive cryptodiran posteroventral floor of the middle ear, exposing the prootic in ventral view. *Kayentachelys* also retains features that are primitive for turtles such as palatal teeth, an interpterygoid vacuity, a dorsal process on the epiplastron, and a ninth costal bone in the carapace. *Kayentachelys* thus shows that cryptodires evolved their distinctive trochlear pattern early in

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