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Congenital anomalies in the baboon (*Papio* spp.)

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Abstract

Background—A comprehensive survey of the prevalence of congenital anomalies in baboons has not been previously reported. We report the congenital anomalies observed over a 26-year period in a large captive baboon colony.

Methods—A computer search was performed for all baboon congenital anomalies identified at necropsy and recorded on necropsy submissions.

Results—We identified 198 congenital anomalies in 166 baboons from 9,972 necropsies (1.66% of total necropsies). The nervous, urogenital, musculoskeletal, and cardiovascular systems were most commonly affected. The most common organs affected were the brain, bone, heart, testicle, kidney, penis, aorta, and skeletal muscle. The most frequent congenital anomalies were blindness, seizures, and hydrocephalus.

Conclusions—The baboon has an overall frequency of congenital anomalies similar to humans and other nonhuman primates. Although the most frequently affected systems are similar, congenital anomalies involving the digestive system appear to be less common in the baboon.

Keywords

nonhuman primate; pathology; spontaneous disease; natural

Introduction

A congenital anomaly is any pathological or traumatic discontinuity of tissue, or loss of function of a part, that is present at birth, regardless of the cause. Congenital anomalies may or may not become clinically apparent until years after birth. It is estimated that 2 to 3% of humans are born with a major congenital anomaly, defined as a anomaly having either cosmetic or functional significance [3,16].

Nonhuman primates are valuable animal models for the research of human diseases because they are so closely related to humans, and thus share many fundamental similarities in physiology, anatomy, and genetics [10]. The baboon has been extensively used as a model for human disease [32], including teratology research [9].

Many comprehensive surveys of human congenital anomalies have been published [1-3,13,18,21,22,27]. There are also reviews of congenital anomalies in nonhuman primates [8,11,24,26,34]. Many of these include small numbers of animals and often combine data from several species.

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Although and there have been several reports of individual baboons with chromosomal anomalies [10,19,20] and congenital anomalies, many contained in an excellent review by Hendrickx and Petersen [9], only three surveys of baboon congenital anomalies have been published. One did not state the number of baboons examined or number of congenital anomalies identified [17]; the other two describe a total of three malformations from 436 necropsies [6,7]. A comprehensive survey of the prevalence and variety of congenital anomalies in baboons has not been reported.

The Southwest National Primate Research Center, located at the Southwest Foundation for Biomedical Research (SFBR), houses the largest captive baboon colony in the world. It is an ideal place to study congenital anomalies in baboons. We report the congenital anomalies observed over a 26-year period in this colony.

Materials and Methods

The SFBR baboon colony population averaged approximately 3,250 animals during the 20-year period. Baboons were housed in two 6-acre outdoor corrals, outdoor metal and concrete gang cages, and indoor-outdoor cages. For variable periods of time, baboons were housed inside buildings individually or in groups for research or other special purposes. The baboons ate a diet of commercial monkey chow supplemented with grains, fruits, and vegetables. Water was supplied *ad libitum*. All animal care and procedures were approved by the Southwest Foundation for Biomedical Research Institutional Animal Care and Use Committee.

Baboons that died or were euthanized were necropsied, and appropriate tissue samples were taken for histologic evaluation. Tissues were fixed in 10% neutral buffered formalin, processed conventionally, embedded in paraffin, cut at 5 microns, stained with hematoxylin and eosin or other stains as indicated, and evaluated by light microscopy. When indicated, individual tissues were frozen in liquid nitrogen, stored at −80°C, fixed with 2% glutaraldehyde for electron microscopy, placed in normal saline or transport medium for cytogenetic evaluation, cultured for bacteria and viruses, or frozen in optimal cutting temperature (OCT) compound for frozen sectioning and further evaluation using immunohistochemistry. The necropsies were performed and histopathologic diagnoses were made by board-certified veterinary pathologists. If deemed necessary, cases were referred to the Armed Forces Institute of Pathology (AFIP) or other individual pathologists with specialized expertise for consultation.

A computer search was performed using an internal anatomic pathology database (apath) for all congenital anomalies in baboons. The original medical records, gross necropsy reports, and histopathology reports were retrieved and reviewed as needed for confirmation or clarification. The animal's age, sex, body system, and organ affected were recorded for each congenital anomaly. For cases with more than one congenital anomaly, each anomaly was counted separately with its corresponding system and organ. The percent of population affected was calculated as the number of animals with anomalies divided by the total number of necropsies.

Diagnoses of atelectasis or congestion were not included. Diagnoses of stillbirth were also not included; however, any congenital anomaly seen in a stillborn or aborted fetus was included. Ovarian and renal cysts were only included if they were determined to be congenital by the pathologist performing the necropsy and histologic evaluation. Pituitary cysts are commonly seen in baboons [29] and are typically due to remnants of Rathke's pouch; these were excluded to be consistent with other published surveys. Blindness was included with the central nervous system due to the lack of observed eye abnormalities and

the frequent occurrence of blindness and seizures in the same animal. The data includes the anomalies encountered in three previously reported chromosomal anomalies (trisomy 16, trisomy 17, and trisomy 18) [10,19,20].

Results

We identified 198 congenital anomalies in 166 baboons from 9,972 total necropsies (1.66% of total necropsies). Table 1 summarizes the congenital anomalies by system and prevalence in the overall population. Table 2 lists the congenital anomalies by system and organ, the relative frequency of the anomalies, age at diagnosis and sex.

The most commonly affected systems in decreasing order (number, percent of population, percent of all congenital anomalies) were the nervous (*n*=107; 0.90%; 54.0%), urogenital (32; 0.32%, 16.2%), musculoskeletal (22; 0.22%; 11.1%), and cardiovascular (14; 0.14%, 7.1%), followed by the hematopoietic-lymphatic, digestive, integumentary, respiratory, and special senses. There was also one congenital neoplasm that affected multiple organs and for which a cell of origin could not be determined.

The most common organs affected in descending order (number, percent of congenital anomalies) were the brain (*n*=107, 54.0%), bone (13, 6.6%), heart (9, 4.5%), testicle (8, 4.0%), kidney (7, 3.5 %), penis (7, 3.5%), aorta (5, 2.5%), and skeletal muscle (5, 2.5%).

The most frequent anomalies in descending order (number, percent of congenital anomalies) were blindness (*n*=43, 21.7%), seizures (34, 17.2%), hydrocephalus (25, 12.6%), hematopoietic/lymphoid hypoplasia (8, 4.0%), hypospadias (6, 3.0%), aortic aneurysm (5, 2.5%), cryptorchid (4, 2.0%), and pectus excavatum (4, 2.0%). Seventeen baboons had both blindness and seizures.

Discussion

We report 198 congenital anomalies in 166 baboons, from 9,972 baboon necropsies performed over a 26 year period. This is an overall rate of 1.66%, slightly less than the 1.87% to 3.24% reported in humans [1-3,18,21]. We expected the observed percentage of congenital anomalies in our colony to be less than that of humans and suspect that the true number of congenital anomalies in baboons is actually greater than our findings indicate. The remains of stillborn and aborted fetus are often cannibalized in socially housed groups, which would lead to undercounting of congenital anomalies within these groups [28]. Based on this, it is likely the actual rate of congenital anomalies in baboons is somewhat higher than we report and closer to that in humans.

The frequency of congenital anomalies is less extensively documented in nonhuman primates. This is a result of evaluations in smaller populations and the combination of species in several surveys. The frequency of congenital anomalies has been reported as 0.9 to 1.02% in rhesus macaques [11,26], 0.3 to 1.62% in cynomolgus macaques [11,26], 1.5% in African green monkeys, 1.55% in stumptail macaques [11], and 0.48 to 1.6% in baboons [6,7,17]. Our findings are consistent with those in other nonhuman primate colonies.

In a review of 19 studies of congenital malformations in humans [21], the overall frequency of congenital anomalies was similar, but the most commonly affected systems and organs differed. These differences are believed to be due to variations in methodology and demographic groups [21,23]. Generally, the central nervous and musculoskeletal systems were most frequently affected, followed by the digestive system and then the urogenital and circulatory systems [1,3,18,22]. In our study, the nervous system was the most often affected, followed in descending order by the urogenital, musculoskeletal, cardiovascular,

hematopoietic-lymphatic, and digestive systems. Due to the variability in the studies of human congenital anomalies, it is difficult to make specific conclusions. Generally, the distribution of systems affected appeared similar to humans, although the urogenital system was more frequently involved and the digestive system was less frequently involved in the baboon.

Most previous reports of congenital anomalies in nonhuman primates lacked sufficient cases to evaluate the distribution by system. Peterson et al. evaluated congenital anomalies by body systems in 390 rhesus macaques and 965 cynomolgus macaques [26]. The most frequently affected systems in the rhesus macaque were the musculoskeletal, cardiovascular, nervous, and urogenital systems. In the cynomolgus macaque, congenital anomalies were observed only in the musculoskeletal and cardiovascular systems [26]. While smaller in scope, these data are similar to the findings in our baboons in that the digestive system appears to be underrepresented compared to the reports in humans.

In addition to the overall frequency of congenital anomalies, we observed several individual findings that suggest areas of similarity between the SFBR baboon population and humans that may warrant further study.

Over 40% of the baboons with congenital blindness also had seizures; a cause of the association is unknown. Although occipital seizures constitute 8% of total seizures in a population of humans with epilepsy[5], epilepsy in baboons is predominantly generalized in characterization, and is therefore not associated with focal pathology [14,29]. While many of the epileptic baboons are photosensitive, spontaneous seizures do not appear to originate occipitally [31](Szabó CÁ, personal communication). Blindness is very rare in most baboons with seizures, as tested by visual tracking in conjunction with EEG experiments [31].

Blindness was diagnosed on the basis of clinical observation and history, and not by detailed ophthalmological testing. It is possible that some of the animals had absence seizures, which cause a vacant stare and inattentiveness, with decreased responsiveness to external stimuli[25]. While absence seizures are brief, some animals may have such frequent absences that the unresponsiveness may be prolonged.

It appears likely a genetic component was involved because both conditions appear to have been eliminated by culling sires and dams of affected animals. The SFBR colony is known to have a high frequency of seizures, with a 20% prevalence in the adult animals in the pedigree colony[30]; however, it has been over 4 years since congenital blindness or congenital seizures have been observed in this colony.

Hypospadias occurs when the fragile process of penile and urethral development does not occur correctly. It results in a defect in the development of the ventral aspect of the penis and an ectopic opening on the urethral meatus. It is the second most common genital malformation in newborn human males, after cryptorchidism [12]. Hypospadias and cryptorchidism were also the most common genital malformations in our baboons. Hypospadias was previously reported from this baboon colony [33].

Sinus of Valsalva aneurysms are rare cardiac anomalies which involve the aortic root and may be acquired or congenital. The congenital aneurysm is more common than the acquired form, with an incidence ranging from 0.1 to 3.5% of all congenital heart defects [4]. Aortic aneurysms were the most common congenital cardiovascular anomaly in our baboon colony, and all five involved the root or most proximal aspect of the ascending aorta. Insufficient detail was present in the records to determine if these aneurisms specifically involved the sinus of Valsalva. However, all five had ruptured, with four of five hemorrhaging into the

pericardial space consistent with a heart base location. Although the frequency of aortic aneurysms is much less than reported in humans, it appears the baboon may serve as a suitable model.

Pectus excavatum is the most common congenital abnormality of the chest wall in humans, accounting for 90% of all congenital chest wall abnormalities [15]. The deformity does affect both sexes in humans, but is markedly more common in males, with a ratio of 5:1 or higher [15]. Pectus excavatum was also the most common congenital chest wall anomaly in our baboons and all four affected baboons were male. An atrial septal defect is an abnormal opening in the atrial septum that allows communication of blood between the left and right atria [16]. It is the most common congenital cardiac anomaly in humans [16] and in these baboons.

The baboon has a prevalence of congenital anomalies similar to humans. While sharing many frequently observed congenital anomalies with humans, the baboon appears to have a lower number of congenital anomalies involving the digestive system. Several specific anomalies share features of those seen in humans, underscoring the baboon's potential as an animal model for such conditions.

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Table 1

Congenital anomaly numbers, percent by system, and percent by population

Body System	Number of Anomalies	% of Anomalies	% of Population*
Central Nervous	107	54.0	0.90
Urogenital	32	16.2	0.32
Musculoskeletal	22	11.1	0.22
Cardiovascular	14	7.1	0.14
Hematopoietic-Lymphatic	8	4.0	0.08
Digestive	7	3.5	0.07
Integumentary	3	1.5	0.03
Respiratory	2	1.0	0.02
Special Senses	2	1.0	0.02
Multiple Systems	1	0.5	0.01
Total	198	100.0	

* Animals with multiple anomalies in the same system were only counted once

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Table 2

List of all congenital anomalies with average age and sex

BODY SYSTEM	ORGAN	MORPHOLOGY	#	% of anomalies	Avg Age ± S.D.	Male	Female
NERVOUS			107	54.0		54	52
	BRAIN		107	54.0		54	52
		BLIND	43	21.7	0.7 ± 0.7	24	20
		SEIZURE	34	17.2	1.9 ± 2.6	19	16
		HYDROCEPHALUS[1]	25	12.6	1.4 ± 4.1	9	13
		PORENCEPHALY	2	1.0	0.7 ± 0.6	1	1
		ANOMALY	1	0.5	0.8	1	0
		DYSPLASIA	1	0.5	1.2	0	1
		LEUKODYSTROPHY	1	0.5	0.0	0	1
UROGENITAL			32	16.2		19	13
	TESTICLE		8	4.0		8	0
		CRYPTORCHID	4	2.0	4.2 ± 2.0	4	0
		MONORCHID	2	1.0	4.5 ± 4.5	2	0
		HYPOPLASIA	2	1.0	15.6	2	0
	KIDNEY		7	3.5		4	3
		HYPOPLASIA	2	1.0	8.0 ± 7.6	1	1
		NEPHROBLASTOMA	2	1.0	0.6 ± 0.1	1	1
		CYST	1	0.5	0.0	1	0
		NEPHROMA	1	0.5	0.2	1	0
		PYELECTASIS	1	0.5	0.0	0	1
	PENIS		7	3.5		7	0
		HYPOSPADIAS	6	3.0	3.3 ± 3.2	6	0
		PERSISTENT FRENULUM	1	0.5	10.7	1	0
	UTERUS		4	2.0		0	4
		HYPOPLASIA	3	1.5	16.9 ± 5.0	0	3
		CYST	1	0.5	0.7	0	1

BODY SYSTEM	ORGAN	MORPHOLOGY	#	% of anomalies	Avg Age ± S.D.	Male	Female
	OVARY		3	1.5		0	3
		CARCINOMA	1	0.5	0.6	0	1
		CYST	1	0.5	0.8	0	1
		HYPOPLASIA	1	0.5	6.8	0	1
	OVIDUCT	HAMARTOMA	1	0.5	0.6	0	1
	UMBILICAL CHORD	STRICTURE	1	0.5	0.0	0	1
	VAGINA	HYPOPLASIA	1	0.5	0.0	0	1
MUSCULOSKELETAL			22	11.1		14	8
	BONE		13	6.6		8	5
		PECTUS EXCAVATUM	4	2.0	0.2 ± 0.4	4	0
		HYPOPLASIA	2	1.0	0.5 ± 0.2	1	1
		MALFORMATION	2	1.0	0.4 ± 0.4	1	1
		CLEFT PALATE	1	0.5	0.0	1	0
		KYPHOSIS	1	0.5	7.3	0	1
		MULTIPLE ANOMALY	1	0.5	0.0	1	0
		POLYDACTYLISM	1	0.5	0.0	0	1
		SCOLIOSIS	1	0.5	7.3	0	1
	SKELETAL MUSCLE		5	2.5		3	2
		HERNIA	3	1.5	7.4 ± 10.2	2	1
		MYOPATHY	2	1.0	0.3 ± 0.3	1	1
	TENDON	ARTHROGRYPOSIS	3	1.5	0.1 ± 0.1	2	1
	PERITONEUM	ANOMALY	1	0.5	0.0	1	0
CARDIOVASCULAR			14	7.1		5	9
	HEART		9	4.5		3	6
		ATRIAL SEPTAL DEFECT	3	1.5	0.4 ± 0.3	1	2
		ENDOCARDIOSIS	2	1.0	0.8 ± 0.6	1	1
		CYST	1	0.5	0.0	0	1
		DYSPLASIA	1	0.5	3.3	0	1

BODY SYSTEM	ORGAN	MORPHOLOGY	#	% of anomalies	Avg Age ± S.D.	Male	Female
		PERSISTENT DUCTUS ARTERIOSIS	1	0.5	0.4	1	0
		PATENT FORAMEN OVALE	1	0.5	0.0	0	1
	AORTA	ANEURYSM	5	2.5	0.3 ± 0.1	2	3
HEMATOPOIETIC-LYMPHOID			8	4.0		4	4
	SPLEEN	HYPOPLASIA	3	1.5	0.2 ± 0.2	1	2
	LYMPH NODES	HYPOPLASIA	2	1.0	0.2 ± 0.2	2	0
	THYMUS	HYPOPLASIA	2	1.0	0.2 ± 0.2	1	1
	BONE MARROW	HYPOPLASIA	1	0.5	0.0	0	1
DIGESTIVE			7	3.5		3	4
	LIVER		3	1.5		1	2
		ADRENAL-HEPATIC FUSION	1	0.5	13.2	0	1
		PIGMENTATION	1	0.5	0.0	0	1
		VASCULAR ANOMALY	1	0.5	0.7	1	0
	ANUS	ATRESIA	1	0.5	0.0	1	0
	COLON	MEGACOLON	1	0.5	0.1	1	0
	GALLBLADDER	ANOMALY	1	0.5	0.0	0	1
	PANCREAS	SPLENIC FRAGMENTS	1	0.5	4.4	0	1
INTEGUMENTARY			3	1.5		1	2
	SKIN		2	1.0		1	1
		CHORISTOMA	1	0.5	0.0	1	0
		CLEFT LIP	1	0.5	0.0	0	1
	EYELID	MALFORMATION	1	0.5	0.2	0	1
RESPIRATORY			2	1.0		1	1
	LUNG		2	1.0		1	1
		DYSPLASIA	1	0.5	0.0	1	0
		HYPOPLASIA	1	0.5	0.0	0	1
SPECIAL SENSES			2	1.0		2	0
	EYE		2	1.0		2	0
		COLOBOMA	1	0.5	13.3	1	0

BODY SYSTEM	ORGAN	MORPHOLOGY	#	% of anomalies	Avg Age ± S.D.	Male	Female
		MICROPHTHALMIA	1	0.5	0.0	1	0
MULTIPLE SYSTEMS	MULTIPLE ORGANS	MALIGNANT NEOPLASM	1	0.5	0.8	1	0

[1] Sex was undetermined for three baboons with hydrocephalus .