Experimental animal models in scoliosis research: a review of the literature

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Abstract

BACKGROUND CONTEXT: Many animal species and an overwhelming variety of procedures that produce an experimental scoliosis have been reported in the literature. However, varying results have been reported on identical procedures in different animal species. Furthermore, the relevance of experimental animal models for the understanding of human idiopathic scoliosis remains questionable.

PURPOSE: To give an overview of the procedures that have been performed in animals in an attempt to induce experimental scoliosis and discuss the characteristics and significance of various animal models.

STUDY DESIGN: Extensive review of the literature on experimental animal models in scoliosis research.

METHODS: MEDLINE electronic database was searched, focusing on parameters concerning experimental scoliosis in animal models. The search was limited to the English, French, and German languages.

RESULTS: The chicken appeared to be the most frequently used experimental animal followed by the rabbit and rat. Additionally, scoliosis has been induced in primates, goats, sheep, pigs, cows, dogs, and frogs. Procedures widely varied from systemic to local procedures.

CONCLUSIONS: Although it has been possible to induce scoliosis-like deformities in many animals through various ways, this always required drastic surgical or systemic interventions, thus making the relation to human idiopathic scoliosis unclear. The basic drawback of all used models remains that no animal resembles the upright biomechanical spinal loading condition of man, with its inherent rotational instability of certain spinal segments. The fundamental question remains what the significance of these animal models is to the understanding of human idiopathic scoliosis.

Keywords: Idiopathic scoliosis; Experimental scoliosis; Animal model; Etiopathogenesis; Review

Introduction

Idiopathic scoliosis is a classic orthopedic disorder of which the etiology and pathogenesis remain largely unknown, although upright spinal biomechanics has been shown to play an important role [1–5]. It occurs only in man and is characterized by the fact that in previously healthy subjects, usually girls, a complex three-dimensional deformity of the spine develops during the period of rapid growth, leading to a disturbed self-image and potential back, pulmonary and cardiac complaints in later life.

In an effort to further clarify the etiopathogenesis of idiopathic scoliosis and explore new therapeutic options, researchers have been inducing scoliosis experimentally in animals for more than a century. Von Lesser [6], in 1888, was the first to describe an experimental scoliosis
model. He produced thoracolumbar scolioses in rabbits after unilateral dissection of the phrenic nerve. Since then, numerous experimental procedures leading to a spinal deformity have been reported in various animal models. The wide variety of procedures and species used, plus the varying success rates for a particular procedure when used in different species, makes it difficult to determine its relevance for the clarification of the etiopathogenesis of idiopathic scoliosis in humans.

The purpose of this study is to provide an overview of experimental animal models used in scoliosis research, with emphasis on the characteristics of the most widely used animals. Moreover, this study considers the relevance of these findings to the understanding of the etiopathogenesis of idiopathic scoliosis in man.

Methods

The MEDLINE electronic database was consulted until December 2010 to search for publications on experimental animal models that produced or were designed to produce a scoliosis. The search terms and results are stated in Table 1. Titles and abstracts of the resulting 1,242 citations were scanned and the following exclusion criteria were applied: (1) articles not written in the English, French, or German language; (2) articles not reporting on an intervention that produced or was intended to produce scoliosis; and (3) case reports. This led to the exclusion of 1,121 citations, resulting in a total number of 121 included articles. Procedures were divided into prenatal and postnatal procedures according to MacEwen, [7] and postnatal procedures were categorized into local interventions (eg, osteotomies, nerve resections, and tethering) and systemic interventions (eg, pinealectomy, feeding Latyrismus peas, and prolonged exposure to light).

Results

An overview of the interventions performed in the different species is given in Table 2. The chicken appeared to be the most widely used experimental animal in scoliosis research, followed by the rabbit, rat, and mouse. Additionally, scoliosis has been induced in primates, goats, sheep, pigs, cows, frogs, and dogs. In this sequence, these animal models are discussed below.

Table 1 Search terms and results

<table>
<thead>
<tr>
<th>Search Query</th>
<th>Number of publications</th>
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<tr>
<td>#1 Scolio</td>
<td>15,251</td>
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<tr>
<td>#2 (((experimental) OR experimental model) OR experimental setup) OR animal) OR animal model) OR in vivo</td>
<td>5,829,832</td>
</tr>
<tr>
<td>#3 Search #1 AND #2</td>
<td>1,242</td>
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* Indicates truncation of the search term.
Because several groups [10,14,15,19,20] reported scoliosis to occur only in about 60% of their pinealectomized chickens, Beuerlein et al. [9,21] investigated whether other aspects of the surgical procedure played a role in the etiology of the disorder. Different resection techniques (cut high, cut low, suction, pull with forceps, and solely cutting the stalk but leaving the gland behind) and the effects of damage to the cerebral cortex were investigated. These experiments revealed that cutting the pineal stalk is the critical stage of pinealectomy after which scoliosis may develop, and not the removal of the gland or some artifact of the surgery [9,21].

The same group also succeeded in producing experimental scoliosis in 15% of normal chickens by keeping them in an environment consisting of intense continuous light [22]. As melatonin production is inhibited by light, decreased serum melatonin levels were observed in these chickens. Light therapy in combination with a pinealectomy increased scoliosis.
incidence from 50% to 80%. It was hypothesized that a threshold level of serum melatonin below which scoliosis may develop was responsible, in conjunction with a factor yet to be identified. These findings, however, could not be reproduced by Cheung et al. [23].

Recently, it has been demonstrated that tamoxifen and trifluoperazine (both calmodulin antagonists) effectively reduced the rate and magnitude of scoliosis in pinealectomized chickens [24]. Because melatonin also acts as a calmodulin antagonist, it has been postulated that loss of this antagonistic effect may be the cause of scoliosis.

Local interventions. Rib transection in chickens did not lead to any marked scoliosis, whereas rib resection did [25]. This led to experiments in which progressive scoliosis in pinealectomized chickens was successfully treated by rib resection on the concave side of the curve [26,27].

The rabbit

No publications concerned prenatal and systemic interventions performed in the rabbit. On the other hand, a wide variety of postnatal local interventions were performed in this species.

Postnatal procedures

Local interventions. Many investigators into experimental scoliosis have emphasized the lordotic nature of scoliosis [28–30], in accordance with the well-known fact that human scoliosis is always a lordotic deformity. This concept has been put forward by several authors [28–32].

In 1952, Somerville [28] produced a progressive structural scoliosis in a growing animal by tethering the spine of rabbits into a lordosis. He approximated spinous processes with sutures, and to reduce posterior spinal growth, he performed laminar cautery. This led him to describe scoliosis as a “rotational lordosis.” Others, however, failed to produce scoliosis using the same method unless a contralateral release of the paraspinal muscles using cauterezation was added [33–35]. However, in a subsequent study by Smith and Dickson [36], it was shown that in animals with severe progressive deformity followed by this procedure, there was localized spinal cord damage because of the cauterezation procedure. They suggested that neural damage instead of muscle release or laminar growth disturbance was responsible for the rapidly progressive deformity.

To investigate the effect of forced lordosis without possible influence of any surgical trauma, Poussa et al. [37] used an external splint to force lordosis in the thoracolumbar junction. This led to a scoliosis in more than half of the animals. These results supported the view that lordosis is a prerequisite for the development of scoliosis in the rabbit. Carpintero et al. [38] elaborated on this asymmetric lordosis theory. They theorized that unilateral dominance of the paravertebral musculature produces an asymmetrical lordosis when erector muscles act behind the anterior axis of flexion of the spinal column. Thus, they performed a unilateral surgical tether between the spinous process apophysis and transverse apophysis at three upper levels of the spine (Figure 2, Left). All surviving animals exhibited a moderate (27°–42°) scoliosis with the convexity opposite to the operated side, including axial rotation.

Hakkarainen [39] simply immobilized growing rabbits in a scoliotic position using a three-point plaster of Paris corset during 2 to 5 weeks. When the initial curve exceeded 30° at the time of relief from immobilization, a progressive scoliosis developed [39]. Subcutaneous scapula-to-contralateral pelvis tethering surgery (Fig. 2, Right) also consistently produced scoliosis in immature rabbits featuring vertebral wedging when the tether was intact for 8 weeks [40]. Recently, electrodes delivering a constant of 50 mA were implanted into one-half of the growth plate of the rabbit spine leading to asymmetric growth inhibition of the vertebral end plate, hence creating scoliosis [41].

Langenskiöld and Michelsson [42–44] performed several surgical procedures in rabbits that turned out to be milestone experiments in scoliosis research. Most of their procedures, however, did not produce scoliosis: sectioning of the phrenic nerve (contrasting Von Lesser [6]), several transpositions of rhomboid muscles, electrocoagulation of anterior costal growth zones, and excision of the costotransverse muscles. Excision of the erector spinae muscle and section of ribs VI to XI, including corresponding intercostal nerves and arteries, only produced slight-to-moderate scoliosis in a limited number of animals.
However, severe scoliosis in all animals was induced after unilateral rib resection (Ribs VI–XI) [43]. In 1962, Langenskiöld and Michelsson [44] reported extensively on their experiments involving rabbits and pigs following up on their previous work. A scoliosis of 50° to 115° developed in approximately 50% of the experimental animals after sectioning of the ligaments around the proximal fourth and fifth ribs (leaving the ligamenta tuberculi costae intact) or sectioning of all ligaments around the costovertebral joints. Hemilaminectomy (including excision of the articular processes) on the right side of five thoracic vertebrae produced scoliosis in all surviving rabbits, of which some progressive (50°–180°). It was postulated that the posterior costotransverse ligament plays an important role in the development of scoliosis by aforementioned techniques.

Sevastik et al. [45,46] osteotomized four ribs in the rabbit, made them override by 1 to 2 cm, and then secured them with wire. This only resulted in a progressive scoliosis when the procedure was combined with simultaneous osteotomy of the opposite ribs. This observation prompted the hypothesis that the spinal deformity in idiopathic scoliosis could initially be because of asymmetric longitudinal growth of the ribs. Support for this was acquired by the fact that a left convex scoliosis developed after intercostal nerve resection on the right side [44,47,48]. According to the authors, this was because of an increase in the vascularity of the structures on the denervated side of the thorax and not to paresis of the trunk muscles [49,50]. Sevastik et al. [51–53] additionally succeeded to correct such experimentally induced scoliosis either by rib elongation with a metallic expander or further resection of intercostal nerves on the opposite side.

In 1961, Liszka [54] postulated that inappropriate afferent impulses to the spinal cord may be the initiating cause of scoliosis. His experiments have been repeated and expanded by other workers [7,55,56]. Laminectomy of four or five segments in 10-day-old rabbits and division of the dorsal roots on one side were reported to produce a scoliosis with convexity toward the side of the divided nerve roots in about half the animals. Both Liszka and MacEwen assumed that severance of the reflex arc was responsible for the development of the scoliosis. However, Alexander et al. [57] asserted that scoliosis produced by rhizotomy in rabbits is not because of interruption of the sensory feedback system, but to simultaneously occurring damage to the anterior horn cells and subsequent motor paralysis as a result of the surgical procedure. Indeed, unilateral lesion of the dorsal column and posterior horn of the spinal cord by coagulation with laser stereotaxic microcoagulation and longitudinal electrocoagulation induced scoliosis in approximately half of the operated rabbits [58,59].

**The rat and mouse**

**Prenatal procedures**

Rats were used to test several chemical agents that are known to be teratogenic in the chicken, in a mammalian embryo. Congenital vertebral deformities were observed...
after application of most tested chemicals. Scoliosis was observed after administration of 6-aminonicotinamide [60]. Also, exposure of pregnant mice to carbon monoxide on gestation day 9 produced offspring with congenital scoliosis [61,62].

Postnatal procedures
Systemic interventions. All the rats supplied with a diet containing 50% of sweet pea (Lathyrus odoratus) seeds developed scoliosis as a result from osteoporosis and collapse of several vertebrae [63–66]. Ponseti et al. [64,65] reported that sweet pea seeds appear to contain a toxic factor (beta-aminopropionitrile) that affects the formation of organic bone matrix, aorta, and possibly other mesodermal structures.

Experimentally induced hemiparkinsonism by unilateral injection of 6-hydroxydopamine into the left area ventralis tegmenti in rats produced a scoliosis with thoracolumbar curves oriented toward the lesion side. The severity of the scoliosis was directly associated with a decrease in striatal dopamine [67]. Also, damage to particular brain stem nuclei related to postural control and equilibrium resulted in scoliotic deformities in rats. This was confirmed by electromyography, which showed imbalance of the paraspinal muscles [68]. O’Kelly et al. [20] used immature rats and hamsters (phylogenetically more closely related to humans than chickens and rabbits) in their experiments to evaluate scoliosis development after pinealectomy. However, scoliosis was not observed.

Bipedal ambulation in addition with systemic interventions. At the same time as O’Kelly et al. [20], Machida et al. [3] demonstrated that scoliosis did not develop in rats after pinealectomy either. However, when they made pinealectomized rats bipedal, scoliosis did occur. Rats were made bipedal by amputating both forelimbs and tail at 3 weeks of age. Subsequently, the rats were stimulated to walk upright by gradually raising food and water. Bipedal rats with only a sham operation (insertion and withdrawal of the needle without application of suction) did develop a cervicothoracic lordosis, but no scoliosis [69].

Bipedal ambulation in melatonin-deficient knockout mice induced scoliosis at a rate of 64.3%. Bipedalism in control mice resulted in scoliosis at a lower rate (25%). Moreover, pinealectomy combined with bipedal ambulation in normal mice produced scoliosis at a rate (70%) similar to what was seen in melatonin-deficient mice [70,71].

Recently, it has been shown that tamoxifen favorably changes the natural history of scoliotic deformities in bipedal melatonin-deficient mice [72]. Also, a higher percentage of scoliosis in bipedal versus quadrupedal rats was observed in rats with induced osteolathrysm by admission of semicarbazide [73].

Local interventions. Right lateral curvatures in growing rats were produced by suturing the inferior angle of the scapula to the ipsilateral pelvis [74]. This led to several morphological and histological changes characteristic of human scoliosis.

Mente et al. [75] applied an external fixation apparatus earlier developed by Stokes et al. [76] to the rat tail to assess if scoliosis progression could be explained in terms of mechanical forces causing vertebral wedging. They not only produced vertebral wedging and scoliosis but also showed that intervertebral discs underwent remodeling and reversal of an induced vertebral wedging by distraction instead of compression [75,77]. This was consistent with the concept of mechanically provoked progression of scoliotic deformities according to the Hueter-Volkmann law [76]. Additionally, vertebral diaphyseal remodeling (Wolff’s law) has been shown to contribute to the deformity in older rats [78]. Stokes et al. [79] also showed that the imposed reduced mobility caused by the use of this apparatus is already a major source of disc changes. They suggest that this may be a factor in disc degeneration in scoliosis.

Primates
Postnatal procedures
Systemic interventions. Cheung et al. [80] performed pinealectomy in 18 rhesus monkeys of which 10 showed total loss of melatonin secretion, but no scoliosis developed in any of the monkeys.

Local interventions. For reasons that remain unclear, procedures that were successful in producing scoliosis in quadrupeds seemed to fail in primates. Unilateral resection of the rib heads, excision of intercostal nerves, division of costotransverse ligaments, excision of the erector spinae muscle, resection of the sacrospinalis muscle, division of the interspinous and interlaminar ligaments, various muscle resections, and denervations have been attempted with little or no success [81]. Contrasting unilateral rib resection, bilateral resection of ribs, and costotransverse ligaments did produce severe scoliosis in monkeys [82]. Scoliosis also incidentally occurred in a series of monkeys during routine virulence testing by intraspinal injection for poliomyelitis vaccines [83]. In addition, scoliosis developed in monkeys after rhizotomy convex to the damaged side in which the severity was dependent on the number of nerve roots cut [84].

Goat, sheep, pig, cow, frog, and dog

Besides the chicken, rabbit, rat, and mouse, other animals were used to a much lesser extent as experimental models in scoliosis research.

Prenatal procedures
Experiments were performed on lambs in which scoliosis was surgically produced in utero [85,86]. Also, pregnant sheep were infected with Toxoplasma gondii [87] or Akabane virus [88] leading to congenital scoliosis. A
possible vestibular origin for scoliosis was recently tested in the frog by unilateral removal of the labyrinthine end organs at larval stages [89]. Microcomputed tomography scans of the skeleton of the adult frogs showed curvature of the spine in the frontal and sagittal planes, a transverse rotation along the body axis, and substantial deformations of all vertebrae.

Postnatal procedures Local interventions. Beguiristain et al. proposed that asymmetrical growth in the neurocentral cartilage of the vertebrae could lead to vertebral rotation and thus to scoliosis. Because in pigs the neurocentral cartilages of the thoracic spine are active beyond the age of one, pigs were selected to undergo unilateral epiphysiodesis of the neurocentral cartilage at four or five vertebral levels consistently produced a structural scoliosis in the pig’s spine with rotation and wedging of the vertebral bodies and convexity on the side of screw fixation [90,91]. This could not be reproduced in a comparable study by Cil et al. [92]. However, by using double pedicle screws, Zhang and Sucato [93] did succeed in creating sufficient epiphysiodesis and hence a scoliosis. In addition, they showed in a recent pilot study that double-screw epiphysiodesis not only can lead to a scoliosis but also could be used to reverse scoliosis in an immature pig model [94].

Scoliosis in the pig and goat was also achieved by a posterior asymmetric tether (optionally combined with concave rib cage ligament tethering) to study the safety and efficacy of fusionless scoliosis treatments, measure certain (vertebral) growth modulations, and to test the permeability and remodeling of the vertebral end plate [95–112]. The possible neurological cause of scoliosis and biomechanical analysis of several instrumentations in thoracolumbar scoliosis were tested on cows [113–115]. Scoliosis in dogs was produced after experimentally induced syringomyelia [116].

Discussion

Idiopathic scoliosis is believed to occur exclusively in humans [117,118]. Many—sometimes exotic—theories have been developed in an effort to better understand this classical orthopedic disorder. The present study aims to provide an overview of the literature on animal experimental models used in studying the etiopathogenesis of idiopathic scoliosis and to discuss the most widely used animals on this subject.

The chicken appeared to be the most frequently used animal in scoliosis research. This is remarkable considering the fundamental differences between the chicken and man in terms of anatomy, biomechanics, and phylogenetical distance to man. The chicken’s spine contains only eight thoracic vertebrae, all lumbar vertebrae are fused, and only two intervertebral spaces (between the sixth and seventh and seventh and eighth vertebrae) have marked intervertebral discs and can be recognized as mobile segments [119]. Despite the fact that the chicken is bipedal, the thoracic and lumbar spine is still mainly horizontally orientated and by no means biomechanically loaded in a manner similar to the human erect spine [2]. Moreover, differences in bone structure exist between avian species and mammalian species.

Nevertheless, pinealectomy in the chicken appeared to be the most frequently used animal model in scoliosis research. This is partly because of the fact that in comparison to mammals, the pineal gland in the chicken is located more superficially (on the dorsal surface of the brain), where it is embedded in a triangular space between the two hemispheres of the telencephalon and can thus be relatively easily approached. Unlike in man, the pineal gland in chickens consists of two parts: a bulb that lies just underneath the skull and a long stalk that attaches the bulb to the posterior aspect of the third ventricle (Fig. 1) [9]. Initial reports commented on anatomical similarities of the scoliotic spine in pinealectomized chickens with idiopathic
scoliosis seen in humans [14,120]. However, more recently also anatomical differences in curve morphology, rib asymmetry, and time of onset of vertebral wedging have been highlighted [16,23,121]. One should, therefore, be cautious when attempting to draw parallels between the pathogenesis of scoliosis in the chicken and human adolescent idiopathic scoliosis [122].

The second most used animal in experimental scoliosis research is the rabbit. The rabbit is a relatively high-level vertebrate, and it is phylogenetically closer to man than the chicken. The vast majority of the studies performed on the rabbit concerned the induction of scoliosis by means of immobilization and operations of the rib cage and spine. It is somewhat surprising that no publications were encountered on pinealectomy performed in the rabbit, given the vast amount of research into this successful procedure in the chicken. Possibly, this could be because of publication bias if the procedure turned out to be unsuccessful.

The rat and mouse, both rodents and essentially nocturnal, are lower level vertebrates compared with the rabbit, dog, and goat. Nevertheless, these species are widely used in orthopedic research because of its low costs and easy handling. Relevant to scoliosis research is that rodents, among many plantigrade mammals, exhibit a natural preference for erect sitting and walking. For these reasons, rats and mice were chosen by Goff and Landmesser [123] to create a bipedal animal model on which gravity acts in a more or less similar manner as it does in men. They amputated the forelegs and tail in newborn rats and mice and stimulated them to walk upright by gradually raising food and water as the animals grew. The bipedal rats rapidly developed an upright bipedal penguin-like waddle, placing each foot alternately before the other, whereas bipedal mice preferred to hop with both feet together [123].

The fact that bipedal rats and mice consistently showed higher incidences of scoliosis in different experimental models compared with their quadrupedal counterparts [3,69–73] clearly shows that the upright posture plays an important role in the origin of scoliosis. Apparently, biomechanical loads acting on the quadrupedal spine protect the spine more or less from developing a rotational deformity. Still, it should be noted that even the spine of bipedal rodents is not loaded in a similar way as the spine of a standing human. Humans are the only vertebrates who are able to ambulate upright with fully extended knees and hips, carrying the weight of their trunk above the pelvis. As a result, only the human spine is subject to posteriorly directed shear loads because of the fact that a considerable part of the spine is posteriorly inclined (Fig. 4) [2]. These particular loads have been shown to render the human spine less rotationally stable [4,124]. In fact, the lack of these posteriorly directed shear loads in all available animal models is their major shortcoming. It requires great effort and rather draconic interventions to create some form of a rotational deformity in any type of animal used, whereas much less is needed in terms of a disturbance of the locomotor, proprioceptive, neuromuscular, or collagen metabolism systems to initiate a compensation into a rotatory deformity in man. In that sense, the development of idiopathic scoliosis could really be the human spine’s preconditioned response to a multitude of offending stimuli, and thus the disorder can truly be called multifactorial.

Obviously, among all other animals, primates are phylogenetically closest to man. From a scientific point of view, this reason alone would make primates the most ideal experimental animal. In addition, most primates spend a considerable part of the day sitting upright or walking in the semierect position. Besides, their vertebral column anatomically closely resembles that of humans. Still, even the primates’ spine is biomechanically not loaded in a manner similar to the human erect spine (Fig. 4). Because of the lack of availability and high costs, the use of primates has been limited. In the few available studies, it was striking that procedures that were successful in producing scoliosis in quadrupeds failed in primates. This raises the principal question of to what extent positive findings in phylogenetically and biomechanically even more distant animals can be translated to humans.

This may be particularly true for the role of melatonin. Pinealectomy and variations on this procedure were subject to extensive research and validation. However, the question remains how the endocrine system in chickens compares to the human situation because a pinealectomy in hamsters, rats, and monkeys did not reveal scoliosis although they are phylogenetically closer to humans [20,80]. So far, studies on melatonin levels in scoliotic patients showed conflicting results [125–127], and melatonin suppletion therapy in patients did not show significant beneficial effects [126].

This review illustrates that the ideal animal model for studying etiopathogenesis of idiopathic scoliosis is neither available in nature nor can it be artificially created in terms of anatomical or biomechanical resemblance to man. Japanese macaques that are trained to walk bipedal might come closest to fulfilling these requirements. If trained to walk bipedally at a juvenile age and over periods of some months or years, they gradually acquire a pronounced lordosis of the lumbar spine [128]. Obviously, the development of such a model is time consuming, not everywhere available and expensive.

After reviewing the vast amount of research on experimental scoliosis, the fundamental question that remains is what the significance of these animal models is to the disorder of human idiopathic scoliosis. Idiopathic scoliosis is a rotatory deformity of the growing spine that occurs in previously more or less normal children. It is striking that because of simple biomechanical conditions, the human spine has been shown to be less rotationally stable than all other spinal constructs in nature, thus making it likely that relatively little effort is needed to induce a progressive rotatory deformity, that is, idiopathic scoliosis [4]. Unfortunately, all historic animal models lack these basic
mechanical prerequisites because they are subject to forces that decrease rather than increase this rotational tendency [124]. Logic dictates, therefore, that rotational equilibrium of the spine is more easily disturbed in humans than in any other vertebrate. In other words, to create a rotational deformity of the spine in any animal requires draconic interventions that are most likely not needed in the human spine. Failure to appreciate this (basic) limitation of all animal models in scoliosis research leads to unwarranted assumptions of severe underlying disorders that, however necessary to produce scoliosis in animals, obviously do not play the same role in humans.

Although our literature search was extensive, this review has some limitations. Articles in other languages other than English, French, or German were not included, and only the MEDLINE database was used. Also, some studies found were of low power because of the limited use of animals. As we decided to exclude articles not reporting on an intervention that produced or was intended to produce scoliosis, articles in which scoliosis was observed in a strain of animals because of inbred or environmental pollution were excluded.

Conclusion

This review shows that scoliosis-like deformities of the spine can be created with various procedures in many animals. The chicken has been most widely studied, followed
by the rabbit, rat, and mouse. Additionally, scoliosis has been induced in primates, goats, sheep, pigs, cows, dogs, and frogs. Most animal models, in particular the widely used chicken, have fundamental shortcomings in areas that have been shown to be particularly relevant in the etiopathogenesis of human idiopathic scoliosis. Especially the lack of a model that biomechanically resembles the human spinal loading condition is a major drawback in scoliosis research. For this reason, in every study, rather draconic interventions were needed to create a rotational spinal deformity in an animal. However, these interventions do not correspond at all to the much more subtle disturbances that can lead to a progressive rotatory deformity in the already rotationally unstable growing human spine. Therefore, the relevance of these animal models for increasing our understanding of human idiopathic scoliosis seems limited.

References


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