Palatal canine displacement: Guidance theory or an anomaly of genetic origin?

A letter to the editor from Adrian Becker, with a response from Sheldon and Leena Peck, and Matti Kataja

The Angle Orthodontist *recently published an article entitled "The palatally displaced canine as a dental anomaly of genetic origin" by Sheldon and Leena Peck and Matti Kataja (1994;64:249-256). These authors argued that genetic factors are the primary etiology of palatal displacement and subsequent impaction of maxillary canines. Following publication, we received a Letter to the Editor from Adrian Becker, who discussed an alternative scenario – the guidance theory – in which the presence of other dental anomalies causes environmental conditions which result in palatal displacement of the canines. Peck, Peck, and Kataja prepared a response to Becker's letter and we present them here together as a single Special Article.*

In defense of the guidance theory of palatal canine displacement

Adrian Becker

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Drs. Peck, Peck, and Kataja are to be congratulated on the thoroughness with which they have reviewed the literature in their effort to present the palatally displaced canine as an anomaly of genetic origin.[1] They have invested an enormous amount of care and concern in searching out the evidence from as many sources as possible, including the French, German, Czech, Korean, Swedish, Finnish, Italian, and Japanese literature, in order to present their case as soundly and as unequivocally as possible. However, having done this, they proceed to draw inferences and conclusions which are unfounded and inappropriate. The same evidence which they present in support of a purely genetic etiology may be used, with no less effectiveness, to argue the case for the guidance theory of canine palatal displacement[2-15] – a case they set out to discredit.

As a believer in many aspects of the guidance theory, I beg to be allowed to use your columns to come to its defense in the face of this broadside attack. In so doing, I shall try to follow the argument in the order and manner in which it has been presented in the text of the article by Peck et al.[1]

There is a considerable body of evidence for considering palatal and facial (buccal) displacement of the maxillary canine as different phenomena. However, this should have been fully referenced in the text as this point is used subsequently as one of the cornerstones of the argument in the debate. This opinion does have its detractors.[16,17]

Peck et al. state that "...palatal displacement...is a positional anomaly."[1] This surely cannot be the case. Clearly observed in both conditions, during both clinical and radiological examination and by direct visual inspection at the time of surgical exposure, the apex of the canine is generally in its normal location, in the line of the arch and close to its ideal a-p position. The only difference is that the long axis of the tooth has become displaced, buccally or lingually, in the coronal direction. This is a clear indication that the early developmental location of both conditions was quite normal. There can be no doubt that the long axis of an untreated tooth accurately indicates, "where it is coming

from," and, accordingly, positional anomaly does not seem to be a logical possibility.

In the case of the buccal canine, Peck et al. appear to concur that the normal buccal eruption path of the canine has been exaggerated (by crowding that exists between the erupted permanent lateral incisor and first premolar), representing a modification of the guidance that Broadbent[18] described, in relation to the development of the normal dentition. Guidance is, therefore, more buccal than distal. In the other case, the protagonists of the guidance theory would postulate that it is equally reasonable to expect certain hereditary factors, such as missing lateral incisors or late-developing roots of a small or peg-shaped lateral incisor, to deprive the relatively early arrival of the canine of this guidance,[2,5-12,14-15] while the presence of excessive space permits it to move palatally.[8]

It would seem that the argument presented by Peck et al. against the guidance theory is being used selectively—a stand which appears to be as illogical as it is untenable.

The authors then proceed to divide the evidence into five separate areas, the first of which relates to the finding of other dental anomalies concomitantly occurring with PDC.

These other dental anomalies are almost certainly genetic in character, but their relationship with PDC could be one of two. Either we must presume premise number 1—the existence of these genetically-determined anomalies has brought about an environmentally-generated alteration in the eruption pattern of the canine, as the guidance theory would hold, or we must presume premise number 2—the PDC is also genetically determined, which is the stand these authors take. However, Peck et al. misinterpret the conclusions from the quoted earlier works. These conclusions are that, "...they noted an interrelationship among observed anomalies of tooth position, number, and size and suggested a shared etiology," but Peck et al. add their own interpretation, with the words, "...without actually recognizing heredity as the basis." By inserting this latter phrase they reject, out of hand, that premise number 1 is a possibility.

Occurrence of other dental anomalies concomitant with PDC

Referring to the work of many independent authors, including that of your humble correspondent, Peck et al. attest to a linkage of these anomalies with PDC. They go on to quote the works of Garn and Lewis and co-workers[19-21] which confirm these associated anomalies as being genetic in nature and linked to one another in their heredity. However, it does not follow that, merely because PDC is found in association with genetically-controlled anomalies, that it is also genetically-controlled. Premise number 1 has not been disqualified and remains a possibility.

At this point, Peck et al. introduce the issue of a totally irrelevant and unrelated anomaly—the phenomenon of the transposed maxillary canine and first premolar (Mx.C.P1). At least, if it were related and therefore relevant, this must first be proven. Without actually saying so, Peck et al. hint that since Mx.C.P1 has a polygenic origin and is also found in association with the genetically-controlled anomalies, a similar origin for PDC may be present.

The contrary would appear to be the case and most of the important clinical signs seem to negate their contention. In Mx.C.P1, not only is the canine crown distally placed, but the root is usually even further distal, often over the second premolar[22] or first molar root apex. If we are to perform the same exercise as suggested earlier, we would find that by following the general direction of the long apex of the two transposed teeth we could determine quite accurately "where the teeth are coming from." In this instance, there is a clear and unequivocal ectopic development of the entire tooth bud from the earliest stages, which indicates a positional anomaly of major proportions. As the authors point out, these cannot be causally-related to anomalous lateral incisors. These cannot, therefore, be labeled as anything but genetically-controlled. Premise number 1 is invalid in this case, but only in this case.

By contrast, PDC usually refers to a crown displacement only, with the root more or less ideally placed. In the day-to-day clinical treatment of palatal canines, experience shows that tipping of the crown into its place in the arch is usually sufficient to achieve good alignment, without, in most cases, resorting to labio-lingual root torquing or mesiodistal uprighting. Clearly then, the canine-first premolar transposition is only a symptomatically-related phenomenon, in terms of the teeth involved, but not in relation to its causal origin. Any etiologic comparison between Mx.C.P1 and PDC is unfounded and misleading.

Bilateral occurrence of PDC

Bilateral occurrence is the second arena on which Peck et al. base their argument. They contend that the high rate of bilateral occurrence points to the existence of genetic factors in the occurrence of PDC. However, the above-mentioned premises number 1 and 2 are both possible candidates as etiologic factors. Premise number 1 could provide the answer that the same associated genetic dental anomalies tend to occur bilaterally and thus produce an environment conducive to the lack of guidance that may lead to PDC.

Sex differences in PDC occurrence

Sex differences show a predilection of PDC for females, and Peck et al. indicate this to the reader in the well-documented literature. However, small, missing, and peg-shaped teeth are also more common among females! Do we deduce that these form the environment that predisposes to PDC or do we assume that the genetic control reroutes the canine, with no influence from the neighboring teeth?

Familial occurrence of PDC

Heredity and familial occurrence are intimately bound up with one another. Evidence for the one may equally be used to prove the other. The reader may like to explain this as polygenic heredity,[23] or as autosomal dominant,[24] or in terms of the guidance theory. In the absence of more definitive evidence, the reader's opinion is as good and as valid as anyone else's.

Population differences in PDC occurrence

Population differences come under scrutiny as the next parameter studied, with the finding that African and Chinese samples appear to be much less affected. Here, however, few reports are found in the literature. The conclusion that, "...palatal displacement of the canine seems to be a predominantly European trait,"[1] may be true, but it does not help in finding the cause. What we would like to know is whether the proven genetic dental anomalies are similarly reduced. By isolating PDC from the other entities, we may provide the key that could offer some hope of deciding the issue. Disappointingly, no such information appears to be available, leaving the reader with both premise number 1 and premise number 2 still viable, and the debate far from resolved.

Critique of earlier hypotheses on PDC etiology

In dealing with mechanical causes, Peck et al. dismiss retained deciduous teeth or space inadequacy as being what they later refer to as, "...the occasional influence of environment and adventitious factors."[1] Nevertheless, they themselves recommend extraction of retained deciduous teeth as a prophylactic measure, quoting four important published references on the subject, of which three are objective studies and one the considered view of an experienced teacher! Furthermore, we may see from the treatment of our patients that, when we mechanically reopen space in the arch for the canine by moving the posterior teeth distally and gathering up anterior spacing (the incisors are usually small and spaced), we may often be rewarded with the spontaneous eruption of the canine, close to its normal position. This scenario may be even more pronounced following the remedial extraction of maxillary premolar teeth in the rarer instances when actual crowding exists.[2,6] It is quite clear that the deciduous canine or the lack of space in these cases is obstruction and displacing (guiding) the canine, since the sudden provision of space would otherwise not permit spontaneous eruption and in an improved position.

This being so, at least some degree of the etiologic credit must be accorded the guidance theory. The fact that Peck et al. recommend prophylactic extraction of deciduous canines suggests that the mechanical theories are not as "...simplistic and inadequate"[1] as they would have us believe.

In the initial part of this discussion, crowding was implicated as the cause of buccal displacement of maxillary canines and this is undoubtedly true for the vast majority of cases that we see in our offices. Nevertheless, there is a small number of cases in which one may diagnose severe buccal displacement, often bilaterally, that is not related to crowding. To the contrary, there may be more than adequate space in the line of the arch for these teeth to erupt into their places. Instead, they may be seen to erupt high, through the oral mucosa, above the attached gingiva. A distance exists between them and the roots of the adjacent teeth, effectively eliminating guidance as a factor. They also seem to exhibit a fairly high relapse tendency, after they have been brought into place. We may be excused if we were to venture to speculate that these canines have developed from ectopically located and buccally directed tooth buds, which places them in the genetic control area and more akin to Mx.C.P1 cases, for the purposes of comparison, than to either the more usual form of buccal canine or to the PDC.

In two separate and distinct works[8,10] quoted by Peck et al., we reported the occurrence of small, peg-shaped, and missing lateral incisors concomitant with palatal canines, in higher frequency than is found in the general population. From these works an important yet largely overlooked fact comes to light: within a random sample of the general population, 4% of the people will have small lateral incisors, 1.8% will have peg-shaped lateral incisors, and 1.3% will have no lateral incisors at all. Of the palatal canine patients within this random sample, the frequency of small and peg-shaped lateral incisors was 7 to 8 times. However, among the congenitally absent incisors, representing a more severe expression of the genetic trend,[19-21] the frequency was only 3 times that seen among the normal cases.

This being so, it would seem that additional factors are at work and cannot be overlooked. Our assessment of the relationship between these factors was outlined in an hypothesis which we de-

scribed in terms of the guidance theory,[8] an hypothesis which provides a logical and reasoned explanation for this unexpected discrepancy. This frequency relationship in PDC cases between small and peg-shaped lateral incisors on the one hand and missing lateral incisors on the other hand represents a small but possibly very significant piece of information. The guidance theory is the only theory to date which is able to account for this apparent paradox.

Summary

From the foregoing debate it will be quite clear that Peck et al. have provided ample evidence that may be used to fuel the arguments of both sides:

1. Their material does not contradict the possibility that environmental factors may give rise to palatal displacement of canines generated by genetic anomaly of the adjacent teeth.

2. The buccally displaced canine finds itself similarly environmentally compromised by the different factor of crowding which leads to its characteristic buccally ectopic guidance pattern.

3. (a) Canines that are transposed with the premolar, (b) others that have erupted ectopically, high in the buccal sulcus and in the absence of crowding, and (c) certain palatal canines whose root apex is located markedly distant from their designated site, may all be labeled as genetically controlled with a fair degree of confidence.

4. In between these clearly defined entities there exists a "gray area" in which it is probable that the etiology of the individual displaced teeth may be linked to a combination of circumstances that obey premise number 1 and premise number 2, to varying degrees.

The guidance theory cannot provide the complete answer to the etiology of the palatally displaced canine. Were this so, we would find PDC every time there was an anomalous or missing lateral incisor. Equally, it may not yet be discounted out of hand and certainly not on the basis of the evidence provided in the article in question.

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References

1. Peck S, Peck L, Kataja M. The palatally displaced canine as a dental anomaly of genetic origin. Angle Orthod 1994;64:249-56.
2. Jacoby H. The etiology of maxillary canine impaction. Am J Orthod 1983;84:125-32.
3. Hitchin AD. The impacted maxillary canine. Br Dent J 1956;100:1-14.
4. Kettle MA. Treatment of the unerupted maxillary canine. Dent Practit 1958;8:245-55.
5. Miller BH. The influence of congenitally missing teeth on the eruption of the upper canine. Dent Practit 1963;13:497-504.
6. Becker A. Etiology of maxillary canine impactions (letter to the editor). Am J Orthod 1984;86:437-438.
7. Moss JP. The unerupted canine. Dent Practit 1972;22:241-8.
8. Becker A, Smith P, Behar R. The incidence of anomalous maxillary lateral incisors in relation to palatally-displaced cuspids. Angle Orthod 1984;54:218-225.
9. Becker A, Zilberman Y, Tsur B. Root length of lateral incisors adjacent to palatally-displaced maxillary cuspids. Angle Orthod 1984;54:218-25.
10. Brin I, Becker A, Shalhav M. Position of the maxillary permanent canine in relation to anomalous or missing lateral incisors: a population study. Europ J Orthod 1986;8:12-6.
11. Oliver RG, Mannion JE, Robinson JM. Morphology of the lateral incisor in cases of unilateral impaction of maxillary canine. Br J Orthod 1989;19:9-16.
12. Zilberman Y, Cohen B, Becker A. Familial trends in palatal canines, anomalous lateral incisors and related phenomena. Europ J Orthod 1990;12:135-9.
13. Brin I, Becker A, Zilberman Y. Resorbed lateral incisors adjacent to impacted canine have normal crown size. Am J Orthod Dentofac Orthop 1993;104:60-6.
14. Bass TB. Observations on the misplaced upper canine tooth. Dent Practit 1967;18:25-33.
15. Becker A. Letter to the editor. Europ J Orthod 1993;15:425.
16. Bjerklin K, Kurol J, Valentin J. Ectopic eruption of maxillary first permanent molars and association with other tooth and developmental disturbances. Europ J Orthod 1992;14:369-375.
17. Kurol J. Letter to the editor. Europ J Orthod 1993;15:425.
18. Broadbent BH. Ontogenic development of occlusion. Angle Orthod 1941;11:223-241.
19. Garn SM, Lewis AB, Vicinus JH. Third molar polymorphism and its significance to dental genetics. J Dent Res 1963;42:1344-63.
20. Garn SM, Lewis AB, Kerewsky RS. Third molar agenesis and variation in size of the remaining teeth. Nature 1964;201:839.
21. Garn SM, Lewis AB. The gradient and the pattern of crown-size reduction in simple hypodontia. Angle Orthod 1970;40:51-8.
22. Peck L, Peck S, Attia Y. Maxillary canine-first premolar transposition, associated dental anomalies and genetic basis. Angle Orthod 1993;63:99-109.
23. Sottner L, Racek J. Stanoveni dedivosti. Model: retencespicaku. Cas Lek Cesk 1978;117:1060-2.
24. Svinhufvud E, Myllarniemi S. Norio R. Dominant inheritance of tooth malpositions and their association to hypodontia. Clin Genetics 1988;34:373-81.

Sense and nonsense regarding palatal canines

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Dr. Adrian Becker and his co-workers at Hebrew University - Hadassah School of Dental Medicine, Jerusalem, have done much over the last dozen years to extend basic understanding of the phenomenon of palatal displacement of the maxillary canine tooth.[1-3] They have been early identifiers of intrinsic differences between palatal and facial displacement of canines. Further, they were among the first to publish data clearly associating palatally displaced canines with concomitant occurrence of other dental abnormalities. Particularly, they documented elevated frequencies of absent and size-reduced maxillary lateral incisors in subjects with palatal displacement of one or both maxillary canines.

Despite these creditable contributions, Dr. Becker and his associates seem also to have invited controversy because of their advocacy of an ambiguous hypothesis to explain certain variations in maxillary canine position. They have propounded more than any of their predecessors or contemporaries the notions that maxillary lateral incisor guidance is a key for normal canine positioning and that anomalies of the lateral incisor are predisposing factors in palatal canine impaction.[1-6] They acknowledge that influences shaping their thoughts came from the observations of Miller[7] and Broadbent[8] recorded 30 to 50 years ago. In 1981, Becker and colleagues[1] described the concept – now labeled "the guidance theory of palatal canine displacement" – in this way:

> There appear to be two processes in the palatal displacement of the maxillary cuspid. The first is a developmental one, related to absence of guidance by the lateral incisor, which opens a new course for a downward path on the palatal side. The second relates to a more advanced period, when the tooth is moving down into a narrower part of the alveolar process. If given the space or only the interference of the deciduous teeth, [the canine] will tend to improve its position and frequently break through the mucosa on the buccal or labial side. It is the presence of permanent tooth roots at this late stage that can prevent the rectifying movement of the cuspid. This would account for the high incidence of peg-shaped (17.2%) and otherwise small (25%) lateral incisors that were found adjacent to palatally displaced cuspids and the relative infrequency [sic] (5.5%) of congenital absence.[1]

Readers who find the description of these processes confusing are not alone.

The article[9] that prompted Dr. Becker to write his present remarks[10] was titled, "The palatally displaced canine as a dental anomaly of genetic origin." It presented a form of meta-analysis of previously published data related to the occurrence of the palatally displaced canine (abbreviated as PDC). Data analysis showed consistent, systematic patterns of PDC occurrence pointing to genetic factors as the primary origin of most palatal displacements and subsequent impactions of maxillary canine teeth.

In his criticism,[10] Dr. Becker chose largely to disregard the quantitative evidence uncovered in our article supporting genetic control as an explanation of best fit for the PDC phenomenon. Instead, he created a "debate" in submitting a "defense of the guidance theory," this concept of local, mechanical interaction between the unerupted maxillary canine and its neighboring lateral incisor that he believes to be the principal cause of palatal displacement of the canine.

In this article, the "guidance theory" will be critically examined – reluctantly, since our PDC studies were *not* about this speculation. Some material from our previous article[9] will be reviewed and additional references will be cited. Readers will have the opportunity to evaluate more evidence supporting a genetic basis for palatal displacement of the maxillary canine tooth.

Problems with "guidance theory" studies

A serious method error appears in the palatal canine studies[1-3,5,6] by Dr. Becker's group. In assembling their samples, "each affected side was [counted] as a separate entity,"[1] rather than using the customary sampling unit of each affected person. Their method artificially inflated total sample sizes (N) and reduced the relative frequencies of the studied anomalies, thus distorting results and making data difficult to compare with the results of others. For example, Brin, Becker and Shalhav[2] reported unusually low prevalence rates for the PDC anomaly, peg-shaped lateral incisors and congenitally absent lateral incisors in a reference sample of 2440 adolescents. Assuming that from their reference sample approximately 30% of individuals with the PDC anomaly exhibited bilateral occurrence (this information was not disclosed in their article), the low prevalence rate of 1.5% for the PDC anomaly found by Brin, Becker and Shalhav[2] actually should have been a rate of 2.4%. In effect, the reported prevalence rate for PDC us-

ing the method of Becker's group represented less than two-thirds (0.63) of its true epidemiologic value. The same kind of error was repeated in the calculations of prevalence rates and statistical tests for the other abnormalities studied in their publications.[1-3,5,6]

Frequency of trait expression is a parameter that Becker and his group studying PDC have failed to reconcile. They found anomalous maxillary lateral incisors in less than half of the individuals with the PDC anomaly.[2] Furthermore, less than 10% of maxillary quadrants with absence or pegged reduction of the lateral incisor were found to have a PDC anomaly.[2] Were these anomalies interrelated mechanically in a cause-and-effect manner —such as control of the expression of the PDC trait through canine misguidance from an anomalous lateral incisor— close to 100% concordance in both directions would be expected. In other words, within the "guidance" model of causality nearly all individuals with PDC would be expected to have a missing or severely reduced adjacent lateral incisor and vice versa. However, according to results of two studies by Becker and co-workers,[1,2] agenesis of the adjacent lateral incisor occurred in 4-6% of PDC cases and conical reduction (peg-shaped laterals) occurred in 13-17% of PDC cases, leaving at least 77% of PDC occurrences unexplained by the "guidance theory." Even with their arguable "small incisor" category making up an additional 25% of the PDC sample, between 52-57% of their PDC cases occurred in the presence of *normal* lateral incisors,[1,2] which again shows the majority of PDC occurrences as unexplained by the "guidance theory" model. In contrast, for dental anomalies under genetic control, variations in gene penetrance and individual thresholds can easily explain the variable phenotypic expression of an abnormality or related abnormalities. Thus, the apparent facts surrounding the relative frequencies of PDC and lateral incisor agenesis/reduction can only be reconciled when PDC, tooth agenesis and tooth-size reduction are recognized to have strong genetic components.

Dr. Becker's experience correcting palatally displaced canines seems to be different from that of many orthodontists. In attempting to strengthen his guidance argument (and to dispute the idea of genetic patterning), he describes the palatal canine as a tooth with a misguided crown and a "more or less ideally placed" root, stating that "tipping of the crown into its place in the arch is usually sufficient to achieve good alignment, without the need to resort to labio-lingual root torquing or mesiodistal uprighting, in most cases."[10] Clinical orthodontists would likely find these generalizations about the biomechanical demands of PDC orthodontic correction highly oversimplified. Dr. Becker's published samples of PDC orthodontic patients[1,5] consisted mostly of children before or around early teen-age, obvious beneficiaries of early diagnosis and usually good candidates for interceptive treatments.[11,12] In many practices, however, a large subgroup of PDC patients is in their middle to late teen-age or older, having been diagnosed late from a chance radiographic finding. Clinical observations with these older PDC patients support the need for surgical exposure and more complex orthodontic movements for the affected canine, in order to achieve good results.[13] Moreover, studies of PDC occurrences in populations with little prospect for receiving interceptive dental services report the average age at diagnosis to be about 18 years, an age at which the finding of bodily palatal displacement of the canine is unambiguous.[14-17] Further evidence supporting total tooth displacement as a feature of most palatal canines is found in the work of Rohlin and Rundquist,[18] studying the positions and anatomy of 65 impacted maxillary canines from 55 patients (mean age=30 years) undergoing autotransplantation procedures. The majority of the canines were palatally displaced (74%) and of these 69% were mesioangular and 54% showed palatal deflection of the root on examination before implantation into newly prepared alveolar sites on the dental ridge. Vertex/true occlusal radiography also has revealed bodily tooth displacement in cases of PDC.[19] These published indications of severe and total malposition of PDC teeth challenge Dr. Becker's bias and point to etiologic factors more profound than can be explained by a mechanical theory of simple crown deflection.

The mechanical cause-and-effect logic that is central to the "guidance theory" has parallels in the history of other clinical misconceptions in dental developmental biology. This thinking earlier had falsely implicated the retained ("resorption-resistant") deciduous canine as a primary *cause* of PDC and canine-first premolar transposition; now the retained deciduous canine in such cases is viewed as a physical *consequence* of the observed anomalies.[9,20] Early explanations of infraoccluded (submerged) deciduous molars present another example of reality and intuition being at odds. Some observers of infraoccluded deciduous molars noted the frequent absence of their permanent successors and they speculated that premolar agenesis was a cause of infraocclusion.[21,22] Later research changed this misconception by showing that the frequency of premolar agenesis under an infraoccluded deciduous molar was not signifi-

cantly different than the agenesis frequency for any premolar in normal circumstances.[23] Infraocclusion and tooth agenesis are now understood to be largely gene-determined abnormalities frequently associated with the occurrence of other dental anomalies, including PDC.[24]

Genetics and the origins of dental anomalies

Despite his denial of the primacy of genetics in the etiology of the PDC anomaly, Dr. Becker seems remarkably comfortable with genetic factors as the cause of other dental disturbances.[10] He does not dispute the genetic origins of maxillary lateral incisor size reduction (pegging) and absence of teeth. He also seems content with the recent finding[20] of genetic origins for another canine positional anomaly, maxillary canine - first premolar transposition. His reluctance to recognize similar evidence[9] supporting genetic causality of PDC is therefore difficult to fathom, except as a personal reaction from his years of association with the now-questioned "guidance theory." In studying families with repeated patterns of tooth malposition, Svinhufvud, Myllärniemi and Norio[25] presented sound evidence that canine malpositioning such as PDC has a genetic component. Part of their discussion is particularly worthy of restatement:

> It is known that an etiologic association exists between hypodontia, small size, malformation and delayed development of teeth. Our study allows the assumption that even certain malpositions of teeth may belong to this spectrum. This is especially reasonable in the case of upper laterals, upper cuspids and second bicuspids because these teeth develop in the critical marginal areas of the dental lamina. One and the same genetic defect in these areas may thus give rise to different phenotypic manifestations.[25]

Gene mediation in most aspects of dental and occlusal development has become a fact of life.[25-28] A mode of genetic transmission often cited in the genesis of dental developmental disturbances is multifactorial inheritance. This type of heritability indeed denotes the presence of important *non-genetic* environmental and individual factors in addition to gene loci, that all interplay in establishing the occlusal and dental phenotype. In other words, individual thresholds and environmental influences, such as prenatal and early postnatal systemic shocks, do figure into the "equation" in attempting an explanation for the phenotypic expression of many dental abnormalities. In occasional instances, obvious local factors have dominant influence, such as cases of early dentofacial trauma adversely affecting the eruptive position of developing maxillary anterior teeth.[29,30] Unlike the relatively clear relationship between early trauma and subsequent tooth ectopia, the "guidance theory" is predicated on a chain of ambiguous events. Therefore, its chances of ever finding a legitimate place as a nongenetic determinant of dental disturbances may be remote without the kinds of clarifications, reproducible studies, and scientific dissection that the genetic model itself has undergone over the years.

An impressive body of evidence is accumulating that links many dental abnormalities together through shared genetic mechanisms.[24-26,31,32] These related dental anomalies include certain types of tooth malposition, such as palatal displacement of maxillary canines. In addition to abnormalities of tooth number, size, and position, disturbances in the chronology and sequence of tooth eruption are reliably thought to be under the same basic genetic control. When such a broad spectrum of abnormalities of teeth is increasingly accepted as having genetic origins, it is unreasonable to assume —as the "guidance theory" does— that PDC is an exceptional phenomenon occurring outside this powerful biological influence.

Near the end of his "defense," Dr. Becker appears to be more conciliatory toward the genetic evidence.[10] In one passage, he describes palatal displacement of the canine as an environmental consequence of "genetic anomalies of the adjacent teeth." He allows that "certain palatal canines...may be labelled as genetically-controlled, with a fair degree of confidence." He still adheres to a tooth-mediated intraosseous guidance mechanism as central to an explanation of the PDC phenomenon. Nevertheless, we find these interpretations moving in a promising direction. Dr. Becker is beginning to think in terms of genetic control. We foresee his group contributing more to an understanding of PDC and other dental abnormalities when they are able to re-evaluate their fine samples in a conventional scientific context, one broader and simpler than "guidance theory" allows.

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References

1. Becker A, Smith P, Behar R. The incidence of anomalous maxillary lateral incisors in relation to palatally-displaced cuspids. Angle Orthod 1981;51:24-9.
2. Brin I, Becker A, Shalhav M. Position of the maxillary permanent canine in relation to anomalous or missing lateral incisors: a population study. Europ J Orthod 1986;8:12-6.
3. Zilberman Y, Cohen B, Becker A. Familial trends in palatal canines, anomalous lateral incisors, and related phenomena. Europ J Orthod 1990;12:135-9.
4. Becker A. Etiology of maxillary canine impactions [letter]. Am J Orthod 1984;86:437-8.
5. Becker A, Zilberman Y, Tsur B. Root length of lateral incisors adjacent to palatally-displaced maxillary cuspids. Angle Orthod 1984;54:218-25.
6. Brin I, Becker A, Zilberman Y. Resorbed lateral incisors adjacent to impacted canines have normal crown size. Am J Orthod Dentofac Orthop 1993;104:60-6.
7. Miller BH. The influence of congenitally missing teeth on the eruption of the upper canine. Dent Practit 1963;13:497-504.
8. Broadbent BH. Ontogenic development of occlusion. Angle Orthod 1941;41:223-41.
9. Peck S, Peck L, Kataja M. The palatally displaced canine as a dental anomaly of genetic origin. Angle Orthod 1994;64:249-56.
10. Becker A. In defense of the guidance theory of palatal canine displacement. Angle Orthod 1995;65(2):95-98.
11. Ericson S, Kurol J. Early treatment of palatally erupting maxillary canines by extraction of the primary canines. Europ J Orthod 1988;10:283-95.
12. Öhman I, Öhman A. The eruption tendency and changes of direction of impacted teeth following surgical exposure. Oral Surg Oral Med Oral Path 1980;49:383-9.
13. Vanarsdall RL Jr. Periodontal/orthodontic interrelationships. In: Graber TM, Vanarsdall RL Jr, editors. Orthodontics, current principles and techniques. 2nd ed. St Louis: Mosby-Year Book, 1994:738-41.
14. Racek J, Sottner L. Contribution to the heredity of retention of canine teeth [in Czech]. Cesk Stomat 1977;77:209-13.
15. Racek J, Sottner L. Our views on the heredity of retention of canines [in Czech]. Sborn Lék 1984;86:355-60.
16. Štefková M, Kamínek M. Position of retained canines and the length of treatment [in Czech]. Cesk Stomat 1979;79:424-30.
17. Kozhokaru MP, Postolaki II. Exteriorization of retained upper canine teeth in adults [in Russian]. Stomatologiia (Moskva) 1980;59:51-3.
18. Rohlin M, Rundquist L. Apical root anatomy of impacted maxillary canines. A clinical and radiographic study. Oral Surg Oral Med Oral Path 1984;58:141-7.
19. Ong A. An alternative technique to the vertex/true occlusal view. Am J Orthod Dentofac Orthop 1994;106:621-6.
20. Peck L, Peck S, Attia Y. Maxillary canine - first premolar transposition, associated dental anomalies and genetic basis. Angle Orthod 1993;63:99-109.
21. Vorhies JM, Gregory GT, McDonald RE. Ankylosed deciduous molars. J Am Dent Assoc 1952;44:68-72.
22. Biederman W. Etiology and treatment of tooth ankylosis. Am J Orthod 1962;48:670-84.
23. Steigman S, Koyoumdjisky-Kaye E, Matrai Y. Submerged deciduous molars and congenital absence of premolars. J Dent Res 1973;52:842.
24. Bjerklin K, Kurol J, Valentin J. Ectopic eruption of maxillary first permanent molars and association with other tooth and developmental disturbances. Europ J Orthod 1992;14:369-75.
25. Svinhufvud E, Myllärniemi S, Norio R. Dominant inheritance of tooth malpositions and their association to hypodontia. Clin Genetics 1988;34:373-81.
26. Garn SM. Genetics of dental development. In: McNamara JA Jr, ed. The biology of occlusal development. Craniofacial Growth Series. Monograph 7. Ann Arbor: Center for Human Growth and Development, University of Michigan. 1977:61-88.
27. Townsend GC, Richards LC, Brown T. Mirror imaging in dentitions of twins — what is the biological basis? In: Brown T, Molnar S, eds. Craniofacial variation in Pacific populations. Adelaide: University of Adelaide, Department of Dentistry, Anthropology and Genetics Laboratory. 1992:67-78.
28. Hu JR, Nakasima A, Takahama Y. Familial similarity in dental arch form and tooth position. J Craniofac Genet Dev Biol 1992;12:33-40.
29. Brin I, Solomon I, Zilberman Y. Trauma as a possible etiologic factor in maxillary canine impaction. Am J Orthod Dentofac Orthop 1993;104:132-7.
30. Peck S, Peck L. Classification of maxillary tooth transpositions. Am J Orthod Dentofac Orthop 1995 (in press).
31. Baccetti T. An analysis of the prevalence of isolated and associated tooth abnormalities in hereditary syndromes: A model for appraisal of genetic control on dental characteristics [in Italian]. Minerva Stomatol 1993;42:281-94.
32. Kurol J. Ectopic eruption and tooth developmental disturbances. In: Davidovitch Z, ed. Biological mechanisms of tooth eruption, resorption and replacement by implants. Boston: Harvard Society for the Advancement of Orthodontics, 1994:371-82.