Otoacoustic Emissions as a Window onto Prenatal Development and Sexual Differentiation

Dennis McFadden, Ph.D.

ABSTRACT

Otoacoustic emissions (OAEs) and auditory evoked potentials (AEPs) exhibit sex differences, and these are present in infants and children as well as in adults. OAEs and AEPs are also different in several special populations of subjects. For females having opposite-sex siblings (OSSZ females) and for homosexual and bisexual females, average OAE measures are shifted towards those of males. Certain AEP measures from homosexual and bisexual females also are masculinized. Certain AEP measures from homosexual males are feminized. These and other facts can be explained by assuming that these special populations received greater than normal exposure to androgens at some point(s) during development, possibly during prenatal development. The implication is that the auditory system may be capable of providing insights into the processes of human development and sexual differentiation.

KEYWORDS: Homosexuality, auditory system, masculinization, hypermasculinization, androgens.

Learning Outcomes: As a result of this activity, the reader will be able to (1) describe the potential impact of high androgen exposure prenatally, and (2) explain various OAE and AEP findings in terms of presumed androgen exposure.

The goals of this article are to provide a summary of a series of experiments from my laboratory along with an explanatory framework that can account for these facts as well as for other related facts. The data to be summarized are from experiments on otoacoustic emissions (OAEs) and auditory evoked potentials (AEPs) in various special populations of subjects.

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Both OAEs and AEPs exhibit sex differences, in newborns as well as in adults. The clear implication is that the physiological mechanisms responsible for the production of OAEs and AEPs were affected differentially in the two sexes at some point(s) in prenatal development. It is not clear what evolutionary advantage might have been associated with these auditory sex differences, and it is fully possible that they are harmless epiphenomena of other mechanisms that were of importance at some time in the history of human evolution. Nevertheless, the existence of such sex differences is interesting and deserving of study, if only because they might lead to insights into other aspects of human behavior.

People working with maladies of the auditory system on an everyday basis might easily come to think of the auditory system as an entity separate from the rest of the organ systems and mechanisms of the body and brain. However, all of those systems and mechanisms must develop together in each individual person, and it would be surprising if there were not cross-effects among organ systems and mechanisms because of this shared developmental history.

A starting point for this review is the assumption that aspects of the cochlear amplifier system are responsible for producing OAEs. Further, because hearing sensitivity is determined in part by the magnitude of displacement of the basilar membrane, it appears appropriate to think about both hearing sensitivity and OAEs as being consequences of, and dependent upon, the cochlear amplifiers. Specifically, when the cochlear amplifiers in an ear are strong, hearing sensitivity is high and OAEs are numerous and strong (see Fig. 1). A positive correlation of just this sort has been demonstrated for OAEs and hearing sensitivity.

Of interest here will be spontaneous OAEs (SOAEs) and click-evoked OAEs (CEOAEs). The measures reported will be average values of SOAEs p.a. ear and average root-mean-square (rms) amplitudes of the click-evoked waveforms. In one study, the correlation between SOAE number and CEOAE amplitude was 0.76, which is in accord with the argument that the mechanisms underlying CEOAEs and SOAEs are overlapping but not identical (by comparison, the correlation between two SOAE measures—number and overall level—was 0.87). Some of the important characteristics of OAEs are shown in Table 1.

Over the years, our procedures for measuring OAEs have become increasingly sophisticated; as a consequence, the average values shown across experiments differ somewhat. Some ways in which our OAE procedures differ from those commonly used in other laboratories are as follows: for both SOAEs and CEOAEs, our subjects lie supine on a small cot; our subjects typically relax in the test room for about 15 minutes prior to any measurements being taken; for CEOAEs, we do not use the differentiation procedures that are commonly used for hearing screening tests, and we use click levels that are weaker than those typically used for hearing screening; for SOAEs, we now collect about 2 minutes of sound from each ear canal and search for SOAE peaks off-line (the algorithm is described elsewhere). Also reported here are AEPs—namely, the auditory brainstem response (ABR), the middle-latency response (MLR), and the long-latency response (LLR). As noted, sex and ear differences exist in certain AEP measures.

<table>
<thead>
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<th>Table 1</th>
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<tr>
<td><strong>Spor</strong></td>
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<td><strong>taneous</strong></td>
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<td><strong>SOAEs</strong></td>
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<td>with Click</td>
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<td>evoked stimulus</td>
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<td><strong>SOAEs</strong></td>
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<td><strong>SOAEs</strong></td>
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<td><strong>SOAEs</strong></td>
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<td>from the ear</td>
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Figure 1 The presumed relationship between OAEs, hearing sensitivity, and the cochlear amplifiers (primarily the outer hair cells of the cochlea).
Table 1 Some Characteristics of Otoacoustic Emissions (OAEs) and Some Sample References

<table>
<thead>
<tr>
<th>The Facts</th>
<th>References</th>
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<tbody>
<tr>
<td>Spontaneous OAEs (SOAEs) are essentially pure tones emitted continuously by most normal-hearing ears.</td>
<td>Kemppälä et al.20 Probst et al.21</td>
</tr>
<tr>
<td>SOAEs are more prevalent in females than males. About 75-85% of females have at least one SOAE compared with 45-65% of males.</td>
<td>Bigger et al.22 Tilmadge et al.23 McFadden and Rasenat24</td>
</tr>
<tr>
<td>Click-evoked OAEs (CEOAEs) are present in essentially all normal-hearing ears and are stronger in females than males. SOAEs are more prevalent, and CEOAEs are stronger, in right ears than left. The sex and ear differences seen in the OAEs of adults are present in newborns as well.</td>
<td>Kemppälä et al.20 Probst et al.21 Tilmadge et al.23 McFadden and Rasenat24 Burns et al.25 Norton27</td>
</tr>
<tr>
<td>Hearing sensitivity is better in females than males, is better in right ears than left, and is better in people having several SOAEs than in people with no SOAEs.</td>
<td>McFadden26 McFadden and Mushah27</td>
</tr>
<tr>
<td>SOAEs appear to be highly consistent through life at least in frequency regions retaining normal hearing sensitivity. SOAEs are more prevalent in people of Asian and African extraction than in people of Caucasian extraction.</td>
<td>Franklin et al.28 Burns et al.29 Prieva et al.30 Whitlead et al.31</td>
</tr>
<tr>
<td>SOAEs are generally quite weak, and most people do not hear their SOAEs. Thus, SOAEs are not the basis for the ringing in the ears (tinnitus) that commonly accompanies hearing loss.</td>
<td>Probst et al.20 McFadden27</td>
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</table>

sures,20 and, just as for OAEs, this is true in infants22 as well as in adults. For these AEP measurements, the subject was seated in a reclining chair inside an electrically shielded, sound-treated room. A shielded insert earphone (Etymotic, model ER-3A) was placed in the left or right external ear canal according to a pseudorandom sequence. The order of data collection was ABR, MLR, LLR. Then the earphone was moved to the second ear, electrode placement and impedance were checked, encouragement was given to the subject, and data were collected for the second ear in the same order as for the first. AEPs were measured using four gold-plated surface electrodes, one on the vertex (Cz), one on the forehead (Fp), and one on each earlobe (A1 and A2). These measurements required approximately 90 minutes to collect. Wakefulness was monitored using the ongoing electroencephalographic waveform. Subjects whose wakefulness could not be maintained by brief conversations were removed from the test booth for short walks. The click levels were approximately 35 and 70 dB HL. The repetition rate was 18.1, 7.1, and 1.1 clicks per second for ABR, MLR, and LLR, respectively. The sweep durations were 20, 120, and 700 ms for ABR, MLR, and LLR, respectively, and 4000, 2000, and 400 sweeps were collected for the three potentials, respectively. The scalp-recorded potentials were bandpass filtered at 0.1–3 kHz, 10–300 Hz, and 1–30 Hz for ABR, MLR, and LLR, respectively. Each sequence of sweeps yielded two waveforms, one for the electrode ipsilateral to the ear containing the earphone and one for the contralateral electrode. Because data were collected with the microphone in both ears, there were four waveforms total for each type of potential for each subject. Estimates of latency and amplitude were obtained from each waveform, and two-way means were calculated as follows. For both latency and amplitude, the left (ipsilateral) value was averaged with the right contralateral value, and the right (ipsilateral) value was averaged with the left contralateral
value (except for ABR wave I, for which all contralateral data were discarded).

**REVIEW OF FINDINGS**

I will first present a series of experimental findings involving OAEs and AEPs and then present a single explanation that appears to be parsimonious, inclusive, and plausible (at least to me).

**OAEs in Females from Opposite-Sex Twin Pairs**

In the process of obtaining estimates of heritability of OAEs from a twins experiment,\(^{11}\) measures also were obtained from opposite-sex dizygotic (OSDZ) twins (by the way, the heritability of OAEs were in the vicinity of 0.75). Of particular interest was the fact that females having male cotwins had OAEs that were displaced toward those of males—that is, their OAEs appeared to be masculinized with respect to those of all other types of females tested.\(^{11,12}\)

Figure 2 provides a summary of the SOAE and CEOAE data obtained in this twins experiment. When the opposite-sex females were compared with the same-sex females, the difference was significant for SOAEs (top panel) and marginally significant for CEOAEs (bottom panel). The OAEs of OSDZ males were not different from those of other males. (The large number of SOAEs in the monzygotic female twins is an interesting finding yet to be further studied or explained. Also yet to be studied are the AEPs of OSDZ females.)

**OAEs in Homosexuals and Heterosexuals**

In another experiment, OAEs were measured in homosexuals and heterosexuals. The mean number of SOAEs and the mean rms amplitude of the CEOAEs are shown at the top and bottom of Figure 3, respectively. Here data are shown separately for the two ears. In accord with several past studies,\(^{11,12}\) OAEs were more numerous and stronger in the right ear than the left, irrespective of sex or sexual orientation. Of more importance here is the fact that the OAEs of homosexual and bisexual females were shifted in the direction of the males—that is, their OAEs appeared to be masculinized compared with those of heterosexual females. These differences were statistically significant.\(^{12}\) Also noteworthy is the fact that heterosexual and nonheterosexual males did not differ in their expression of OAEs. (Bisexuals were not actively recruited for this experiment, but subjects were categorized on the basis of their responses to certain items on a questionnaire administered to all subjects.)

**AEPs in Homosexuals and Heterosexuals**

In a follow-up experiment,\(^{7}\) AEPs were measured in heterosexual and nonheterosexual subjects using two click levels. In all, there were 19 separate measures of latency and amplitude. Five of those measures showed differences between the heterosexual and nonheterosexual females, and five measures showed differences between the heterosexual and nonheterosexual males (with one measure showing differences for both sexes). Not all of these nine AEP measures exhibited a basic sex difference between the heterosexual females and heterosexual males (that is, they appeared masculinized on those measures). In addition, the nonheterosexual males were shifted even further from the heterosexual females than were the heterosexual males (that is, the nonheterosexual males appeared hypermasculinized on those measures). The data in the bottom panels of Figure 4 exhibit a basic sex difference. The top panel illustrates masculinization in the nonheterosexual females, and the bottom two panels illustrate hypermasculinization in the nonheterosexual males. The second panel from the top illustrates a difference between heterosexual and nonheterosexual females in the absence of a basic sex difference between the heterosexual females and males.
Table 2 contains effect sizes for some of the relevant comparisons from the three experiments summarized in the preceding figures. Collins' suggested that effect sizes of 0.2, 0.5, and 0.8 could be considered small, medium, and large, respectively. Medium differences prevail in Table 2.

**DISCUSSION**

Table 3 provides a summary of some of the experimental findings in need of explanation. It is logically possible that different factors are responsible for each of the separate OAE and AEP differences found in the different special
Figure 3  Mean number of SOAEs per ear (top) and mean strength of SOAEs (bottom) for people of differing sexual orientations. Data are shown separately for the two ears. All SOAEs detected between 0.5 and 9 kHz were included here. The differences between the values here and in the twins experiment (Fig. 2) are attributable to differences in procedure. Data were previously published27 and are reprinted here with permission.
exposed to certain noise sources, drugs, or other ototoxic agents than are heterosexual females. Explanations that appeal to lifestyle differences of this sort will be considered first. Then, I will offer a hormonal/developmental explanation that, in its detailed form, appears to account for all of the various OAE and AEP differences noted in Table 3 as well as for additional facts.

**Some Possible Lifestyle Explanations**

Perhaps the primary concern about the auditory differences shown in Table 3 is that the people in these various special populations may have been exposed to one or more lifestyle factors that have produced a hearing loss, either reversible or permanent. Note that the concern here is about an acquired hearing loss attributable to damage to the cochlear amplifiers. As revealed by Figure 1, we would expect there to be a small, inherent difference in hearing sensitivity between any groups exhibiting differences in their OAEs. It is larger differences, perhaps related to differences in lifestyle, that need to be carefully considered when interpreting our results.

Assessment of the possibility of differential hearing loss in the different types of subjects studied led us to take various precautions in all of our experiments. For example, at the time of being scheduled for testing, each subject was told to avoid exposure to intense sounds and various prescription, over-the-counter, and recreational drugs for a period of at least 24 hours prior to the test session. Upon arrival at the test session, the subject was asked about noise exposure and drug use in the past 24 hours, and subjects admitting to having forgotten about the prohibitions were rescheduled. Further, all subjects were given a hearing screening test, and all those not having hearing sensitivity within 20 dB of normal in both ears were excluded from the experiment. A considerable number of subjects were excluded from our experiments because of these precautions. Because hearing screening tests are not capable of detecting small differences in hearing sensitivity, such as those between the sexes or the ears (which requires extensive psychoacoustic testing—see McFadden and Muhraf), it is not possible for us to assert unequivocally that there were no differences in lifestyle-induced hearing loss between our various special popu-
sions and our control groups. However, because we did screen for substantial hearing loss, it seems reasonable to assume that any remaining group differences in hearing sensitivity were small.

Furthermore, the software developed to analyze our SOAE data also allowed a simple, indirect test of the possibility of differential hearing loss in our homosexual and heterosexual subjects. At the heart of this test is the fact that the vast majority of noise- and drug-induced hearing losses progress from high frequencies toward low frequencies. If the diminution in number and strength of SOAEs observed in nonheterosexual females (Fig. 3, where all SOAEs from 0.5 to 9 kHz were included) was attributable to the nonheterosexual subjects having an undetected, acquired hearing loss relative to the heterosexual females, then the effect of that differential hearing loss should be greatest in the high-frequency regions. Accordingly, if all high-frequency SOAEs were excluded from the data analyses, then the differences between heterosexual and nonheterosexual females shown in the top panel of Figure 3 should be reduced or eliminated. However, when only SOAEs below 3 kHz were included, the pattern of the data was the same, and the magnitudes of the relevant differences were even a bit greater (see Table II in McFadden and Passner). (Note that, by contrast with OAES, AEPS should be affected little by small hearing losses.24)

One lifestyle difference between the homosexual and nonheterosexual female groups in our OAE and AEP experiments was in the use of oral contraception (OC). Approximately 40% of the heterosexual females and 14% of the nonheterosexual females were using OC in the two experiments combined. If the use of OC somehow hypermethylated OAES and/or AEPS, then the differences observed in our heterosexual and nonheterosexual females (Figs. 3 and 4) might have been attributable simply to the differential use of OC in the two

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**Table 2** Effect Sizes for Various Comparisons from Three Experiments

<table>
<thead>
<tr>
<th>Measure</th>
<th>NT Females vs. NT Males</th>
<th>OISDD Females vs. SSDQ Females</th>
<th>FHT vs. MHT</th>
<th>FHT vs. FHm + Fbi</th>
<th>MHT vs. FHm + Mbi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of SOAEs</td>
<td>0.57</td>
<td>0.49</td>
<td>0.98</td>
<td>0.57</td>
<td>0.12</td>
</tr>
<tr>
<td>Strength of SOAEs</td>
<td>0.51</td>
<td>0.42</td>
<td>0.96</td>
<td>0.41</td>
<td>0.03</td>
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<tr>
<td>ABR wave I latency</td>
<td></td>
<td></td>
<td>0.12</td>
<td>0.47</td>
<td>0.08</td>
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<tr>
<td>70-db clicks</td>
<td></td>
<td></td>
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<tr>
<td>ABR wave I amplitude</td>
<td>0.59</td>
<td>0.37</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>70-db clicks</td>
<td></td>
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<tr>
<td>ABR wave V latency</td>
<td>0.85</td>
<td>0.62</td>
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<tr>
<td>35-db clicks</td>
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<tr>
<td>ABR wave V amplitude</td>
<td>0.51</td>
<td>0.24</td>
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<tr>
<td>25-dB clicks</td>
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</table>

Calculations based on two-tail means for the OAEs or averages across left and right electrodes and bilateral and contralateral click stimuli for the AEPs. Entries are absolute values. OISDD and SSDQ designate opposite-sex, opposite-sex, heterosexual, and bissexual, respectively. NT, FHm, and M designate nontranssexual, heterosexual, and bisexual, respectively. F and M designate female and male, respectively.

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**Table 3** The Auditory Facts in Need of Explanation

| There are some loud differences in OAES, AEPs, and hearing sensitivity. These sex (and sex) differences exist in newborns as well as adults.
| OAES and AEPS appear to be reasonably stable traits through life. Females from opposite-sex twin pairs have masculinized OAES. Homosexual and bisexual females have masculinized OAES and AEPS. Homosexual males have hypermethylated AEPS but neither hypo- nor hypermethylated OAES.

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Adapted from the original document.
subject groups. To test this possibility, we com-
pared the OAEs and AEPs of users and nonusers of OC (all heterosexual). The result
was that OC use actually masculinized OAEs and AEPs slightly,\(^3\), that the effect was in the
wrong direction for it to be an explanation
for our basic findings. Because of this analysis,
however, the users of OC were omitted from
all analyses of the AEP data.\(^4\)

Related to the question of OC use is the
issue of menstrual effects on the auditory
system. The frequencies of some SOAEs do
shift slightly as a function of the menstrual
cycle,\(^5\) but the effects were so small that, in
our OAE experiment, we took no steps to con-
trol when in the cycle data were collected.\(^6\)
Because some AEP measures were known to
be affected by the menstrual cycle,\(^7\) we
tested our normal-cycling females only during
the last 10 days of their menstrual cycles,\(^8\)
when both estrogen and progesterone levels are
high and when the differences between males
and females are greatest.\(^9\) Thus, an attempt was
made to prevent any group differences in men-
strual cycles from compromising the experi-
mental comparisons.

In summary, then, various elements were
built into the experiments, and a number of
special analyses were performed on the data, in
an attempt to minimize contributions from
possible differences in lifestyle. Of course, in
corroborative research using natural popula-
tions, it would be impossible ever to rule out all
differences in lifestyle.

A Proposed Hormonal Explanation

To review, OAEs and some AEPs exhibit sex
differences (Figs. 2–4). These sex differences
are evident in adults, newborns, and young
children,\(^2\),\(^3\),\(^11\) strongly suggesting that these
differences are the result of mechanisms oper-
ating prenatally and differentially in the two
sexes. Further, OAEs are known to be highly
consistent throughout life,\(^10\),\(^11\) and AEPs also show
high stability across time.\(^12\),\(^13\) Accordingly, it is
plausible that both the differences seen in the
OAEs of OSDZ females and the differences seen in the OAEs and AEPs of heterosexuals
and nonheterosexuals existed at birth. If we as-
tume they did, it becomes reasonable to esti-
mate that the sex differences seen in the OAEs
and AEPs of newborns are the result of the
same mechanism known to be responsible for
numerous other sex differences in body, brain,
and behavior—differential exposure to andro-
gens during prenatal development.\(^14\) That
leads to the viewpoint illustrated in Figure 5.
Greater exposure to androgens during prenatal
development leads to a weakening of the
 cochlear amplifier (which produces a weak-
ing of the OAEs and a small loss in hearing
sensitivity) and also leads to effects on various
auditory nuclei concerned with hearing sensi-
tivity, AEPs, and possibly other auditory char-
acteristics or abilities.

Under this prenatal-androgen-exposure explana-
tion:

1. The cochlear and elements of the auditory
brain of heterosexual females are different
from those of heterosexual males because of
the latter's greater exposure to androgens
during prenatal development. The reason for
emphasizing gestational development is that
the enormous sex difference in androgen
levels present during prenatal develop-
ment disappears at birth, when androgen
levels become the same (and low) in both
sexes.\(^15\) In males, there is a lesser, second
surge of androgens that begins with birth
and persists for about 20 weeks,\(^16\) after
which the androgen levels decline and stay
equal to those of females until puberty.
However, the existence of substantial sex
differences in OAEs and some AEPs at
birth suggests that it is the prenatal expo-
sure to androgens that accomplishes the
preparation of the masculinization of the
auditory system.

2. The cochleas of OSDZ females were
mal
-culturinized because of their greater than nor-
normal exposure to androgens (for females)
during prenatal development, presumably
because of the presence of a male conin in
the shared intrauterine environment. Nu-
merous examples exist of male fetuses ma-
culizing adjacent female fetuses in various
species of litter-bearing mammals.\(^17\),\(^18\) In
Figure 5: The presumed relationship between androgen exposure early in development (possibly prenatal development) and the OAEs, hearing sensitivity, and AEPs seen later in life. The solid lines illustrate the prenatal-androgen-exposure explanation offered here; the dotted lines illustrate commemorations extensions of this explanation.

Humans, the evidence is less uniform. OSDZ females have been reported to be masculinized on measures such as sensation seeking, mental rotation, and dental alignment\(^{19}\) but not on several other measures.\(^{20,21}\) (AEPs have yet to be measured in a group of OSDZ females.)

3. The cochleas, and elements of the auditory brain, of nonheterosexual females also were masculinized by greater than normal exposure to androgens, presumably during certain critical periods of prenatal development. Implicit in this account is the idea that the OAEs and AEPs in our nonheterosexual females were masculinized by the same androgenic mechanisms or processes responsible for the masculinization of whatever brain locations regulate sexual orientation in females.

4. Elements of the auditory brains (but apparently not the cochleas) of nonheterosexual males also received greater than normal exposure to androgens at some point(s) in prenatal development, and as a consequence, some aspects of their AEPs were hypomasculined. The idea of greater than normal exposure to androgens for nonheterosexual males initially appears less intuitive or compelling than the overandrogenization of nonheterosexual females because often when nonheterosexual males are found to differ from heterosexual males, the direction of the difference is toward hypomasculination, not hypermasculinization. Elsewhere\(^{23}\)
I discuss additional evidence for the exis-
tence of both hypomasculinized and hyper-
masculinized characteristics in both female and
male offspring of mothers who received androgen
exposure in utero. The results of this study sup-
port the hypothesis that prenatal androgen expo-
sure has a lasting effect on the development of
the CNS.

Without specifying exactly what the dif-
ferring effects of androgens were at the various
sites involved, or why the androgen exposures
were greater than normal, this prenatal androgen-
exposure explanation does provide a general
overall perspective that appears consis-
tent with all of the auditory results re-
ported previously.

The exploration offered here appeals to
what is commonly called the organizational ef-
fects of hormones — permanent changes ini-
tiated by hormone exposure early in life. For
completeness, it should be mentioned that re-
versible, or activational, effects of hormones
also are known for some auditory measures. As
already mentioned, OAEs, AEPs, and a num-
ber of other auditory measures are affected to
some extent by the menstrual cycle.2,3 Also, the
SCAEs of an adult male taking high levels of
testosterone seemed to become more female-like
as his androgen levels fell.4,5 Essentially noth-
ing is currently known about the relationships
between these activational effects and the ap-
parently organizational effects seen in the spe-
cial populations we have studied to date—
hence their absence in Figure 3.

SUMMARY

My goals for this article were to summarize the
effects of auditory findings and to describe an ex-
planatory framework that seems both to ac-
count for those findings and to fit well with a
number of related facts. In brief, the idea is
that the masculinizations and hypomascu-
lizations seen in the OAEs and/or AEPs of
preterm neonates, nonheterosexual females, and
nonheterosexual males may be attributable to
their having been exposed to higher than nor-
mal levels of androgens at some point(s) in
prenatal development. The implication is that
various auditory structures were masculinized
or hypomasculinized as part of the same pro-
cesses that led to masculinization, hypema-
sculinization, or hyposusceptibility of what-
ever brain structures are responsible for sexual
orientation in humans. If this is correct, it sug-
gests that the auditory system has the potential
to serve as a valuable, noninvasive window
onto the processes of human prenatal develop-
ment and sexual differentiation. In order to ac-
count for all of the various findings about dif-
ferences in body, brain, and behavior in these
special populations, it appears necessary to po-
titute the existence of numerous specific mech-
nisms of androgen exposure, perhaps operating
at different times during development.6 If
the interpretation I am offering for the various
findings reviewed here is correct, the implica-
tion is clear that the auditory system should
dot be viewed as an independent entity devel-
oping in isolation from all the various influ-
ences operating on the other organ systems
and mechanisms of the developing body and
brain. Rather, the processes controlling devel-
opment in the auditory system can be influ-
enced by processes affecting the development
of other organ systems—and presumably the
converse occurs as well.

ACKNOWLEDGMENTS

This article is adapted from a similar, but more
complete, review of the same body of work in
the Archives of Sexual Behavior.7 Plenum
Press, the publisher of that journal, generously
agreed to the publication of this adaptation.
The review, in turn, was based on a presenta-
tion given at the International Behavioral De-
velopment Symposium, May 2000, Minot
State University. A large number of collabora-
tors and assistants have contributed substan-
tially to this work. They are listed by name in the
individual research papers summarized
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butions of E.G. Pasman, J.C. Leschkin, and
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**ABBREVIATIONS**

AIR auditory brainstem response
AEPS auditory evoked potentials
CENOAs click-evoked otoacoustic emissions
HL hearing level
LLR long-latency response
MLR middle-latency response
OAEs otoacoustic emissions
OC oral contraresonance
OSDZ opposite-sex diagnostic
peSPL peak-equivalent sound-pressure level
SOAEs spontaneous otoacoustic emissions

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ARTICLE THREE

SELF-ASSESSMENT QUESTIONS

1. Adult sex differences in otoacoustic emissions
A. are likely due to differential noise-
induced hearing loss in males and females
B. are likely due to the differences in
intra-individual levels of androgens in adults
C. are likely due to differences in androgen
exposure during prenatal development
D. are likely due to the widespread use of
oral contraceptives by women
E. none of the above are true
2. Which statement(s) is/are true?
A. Good hearing sensitivity causes strong otoacoustic emissions
B. Strong otoacoustic emissions cause
good hearing sensitivity.
C. Hearing sensitivity and otoacoustic emissions are independent, unrelated variables.
D. Good hearing sensitivity and strong otoacoustic emissions are both consequences of a common factor.
E. Both C and D are true.

3. Which statement(s) is true?
   A. The otoacoustic emissions and certain auditory evoked potentials of opposite-sex dizygotic females are masculinized.
   B. The otoacoustic emissions and certain auditory evoked potentials of homosexual and bisexual females are masculinized.
   C. The otoacoustic emissions and certain auditory evoked potentials of homosexual and bisexual males are hypermasculinized.
   D. All of the above are true.
   E. Two of the above are true.

4. Which statement(s) is true?
   A. Two of the auditory evoked potential measures showing hypermasculinization for homosexual males were amplitudes.
   B. Not all AEP measures showing differences for homosexuals and heterosexuals also exhibited a basic sex difference.
   C. Otoacoustic emissions are typically stronger in right ears than in left ears.
   D. All of the above are true.
   E. None of the above are true.

5. Which statement(s) is true?
   A. There are no known acertinal effects of hormones on the auditory system.
   B. Menstrual effects are greater on OAEs than on AEPs.
   C. The prenatal-hormonal-exposure explanation offered here also accounts for the large number of OAEs in monosex-gynetic females.
   D. The OAE and AEP differences seen between homosexuals and heterosexuals may be simply attributable to differential hearing loss in the different groups because, to date, no steps have been taken to control for this important variable.
   E. None of the above are true.