

**PERCEPTUAL CORRELATES OF EFFERENT
MODULATION IN THE HUMAN AUDITORY
SYSTEM**

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ABSTRACT

Elicitation of the medial olivocochlear reflex (MOCR) causes a reduction in the amount of gain (amplification) applied by the cochlear amplifier. This gain-control function is thought to play an important role in speech-in-noise perception. Otoacoustic emissions (OAEs) offer a qualitative measure of the effect of the MOCR on cochlear gain, but a quantitative measure is lacking. The aim of this thesis was to test whether any of the putative perceptual correlates of MOCR-induced cochlear gain reduction might provide such a measure.

The first study (Chapter 2) is concerned with the mechanism of the overshoot effect, in which a brief signal presented at the onset of a masker is harder to detect when the masker is preceded by silence than when it is preceded by a “precursor” sound. It has been suggested that, in overshoot, the precursor might reduce cochlear gain by eliciting the MOCR and thereby cause a reduction in suppressive masking of the signal (adaptation of suppression). Overshoot, suppression, and adaptation of suppression were measured in the same participants. While the precursor yielded strong overshoot, and the masker produced strong suppression, the precursor did not appear to cause any adaptation of suppression. Predictions based on an established model of the cochlear input-output function indicate that the failure to obtain any adaptation of suppression is unlikely to represent a false negative outcome. It is argued that overshoot may be due to higher-order perceptual factors such as transient masking or attentional diversion.

Overshoot was therefore not pursued as a quantitative measure of the MOCR.

The second study (Chapter 3) aimed to develop a quantitative measure of the MOCR by modifying the established temporal masking curve (TMC) method for estimating cochlear gain psychophysically. The TMC method involves measuring the lowest masker level needed to just render inaudible a weak signal as a function of the temporal gap between the masker and signal. Here, the masker's duration was shortened so that the masker would not itself elicit the MOCR in time to affect the signal's audibility. A new way of estimating cochlear gain from TMC data by fitting the entire data set with a generic model of the cochlear response function was also developed. Using this approach, the effect on cochlear gain of a broadband-noise elicitor presented to the contralateral ear was measured. The TMCs suggest that the elicitor reduced cochlear gain by 4 dB, on average. OAE suppression measurements in the same participants suggested that this gain reduction was mediated by the MOCR. The approach developed in this chapter provides a quantitative estimate of MOCR-induced cochlear-gain reduction caused by a contralateral elicitor.

The third study (Chapter 4) aimed to assess the validity of recent findings by Yasin et al. (2014), who reported an MOCR-induced cochlear-gain reduction by an ipsilateral elicitor that was four times larger than that found in the second study using a contralateral elicitor. Yasin et al. (2014) estimated cochlear gain reduction using the fixed-duration

masking curve (FDMC) method, which is similar to the TMC method used in Chapter 3. In Chapter 4, the FDMC method was used to estimate the amount of gain reduction caused by a long ipsilateral elicitor, like the one used by Yasin et al. (2014). This was compared to the amount of gain reduction caused by a much shorter ipsilateral elicitor, which was presented at a level that produced the same amount of masking of the signal as the long elicitor, but was too short to have activated the MOCR in time to affect the signal detectability. The long and short elicitors both caused large psychophysical effects, indicating either that the MOCR acts more quickly than previously thought, or that the effect was not due to MOCR-induced cochlear gain reduction. OAE suppression was also found for both the long and short elicitors. It is argued that both the OAE and psychophysical effects of the short and long elicitors may, at least in part, be the result of nonlinear interactions between the elicitor and the masker resulting from direct temporal overlap of their cochlear responses.

This thesis provides evidence against the idea that MOCR-induced cochlear-gain reduction plays a major role in either overshoot or in a recently reported large psychophysical masking effect by an ipsilateral noise, both of which have previously been attributed to the MOCR. This thesis has also contributed towards the refinement of an approach for quantitatively measuring cochlear gain and MOCR-induced cochlear gain reduction by a contralateral noise. In future, this approach could become a valuable audiometric profiling tool, and may give insight into the

individual differences that underlie hearing problems in audiometrically normal listeners. Parametric exploration of the MOCR using this approach may also allow the functional importance of the MOCR in humans to be better understood.

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ABBREVIATIONS AND SYMBOLS

Cochlea nucleus (CN)

Distortion-product otoacoustic emission (DPOAE)

Electromyography (EMG)

Equivalent rectangular bandwidth (ERB)

Evoked otoacoustic emission (EOAE)

Fast Fourier transform (FFT)

Fixed-duration masking curve (FDMC)

Inner hair cell (IHC)

Input-output function (IO function)

Interaural level difference (ILD)

Interaural time difference (ITD)

Lateral olivocochlear reflex (LOCR)

Linear mixed-effects regression model (LMM)

Medial olivocochlear reflex (MOCR)

Middle-ear muscle reflex (MEMR)

Olivocochlear bundle (OCB)

Otoacoustic emission (OAE)

Outer hair cell (OHC)

Peak equivalent (pe)

Root-mean-square deviation (RMSD)

Sound pressure level in decibels (dB SPL)

Standard error of the mean (\pm)

Superior olivary complex (SOC)

Temporal masking curve (TMC)

Tucker-Davis Technology (TDT)

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CHAPTER 1

GENERAL INTRODUCTION

In the middle of the 20th century, descending (efferent) auditory pathways were discovered, some of which terminate inside the cochlea (e.g., Rasmussen, 1946, 1953). Shortly afterwards, it was demonstrated that these efferent pathways can modulate the way in which the cochlea processes sounds (Galambos, 1956). Understanding this top-down modulation of peripheral auditory processing may give insight into dysfunctions such as tinnitus (e.g., Jastreboff, 1990) and hearing impairment (Moore, 2007).

Unlike reptiles and birds, whose efferent pathways project from a single nucleus (e.g., Schwarz et al., 1981; Simmons, 2002), humans and most

other mammals have two distinct peripheral efferent pathways: one that projects from the medial part of the superior olivary complex (SOC) and another that projects from the lateral part of the SOC. The SOC, in turn, receives efferent projections from both primary and non-primary cortical areas (Budinger et al., 2000; Doucet et al., 2002; Coomes and Schofield, 2004) and is the first level of the auditory system at which there is integration of information from the two ears (Caird and Klinke, 1983; Yin and Chan, 1990; Wu and Kelly, 1992). The medial olivocochlear (MOCR) and lateral olivocochlear (LOCR) reflexes operate through the two efferent pathways that project from the SOC to the cochlea. In the cochlea, LOCR fibres terminate on type-I afferent fibres under the inner hair cells (IHCs; Warr and Guinan, 1979; Guinan et al., 1983). The IHCs perform the conversion from basilar-membrane motion to neural firing. In contrast, MOCR fibres terminate on the outer hair cells (OHCs; e.g., Kimura and Wersall, 1962; Spoendlin, 1966; Warr and Guinan, 1979; Guinan et al., 1983), which are involved in amplifying basilar-membrane motion (Davis, 1983; Dallos and Evans, 1995; Jia et al., 2005).

Despite decades of research, the functional purpose of the LOCR and MOCR remains poorly understood. The limited understanding of the LOCR stems largely from the fact that LOCR fibres are thin and unmyelinated, which makes direct recording and electrical stimulation difficult. Two potential functions of the LOCR have been suggested, namely, (i) that it protects the auditory system from damagingly loud sounds (Ruel et al., 2001), and (ii) that it balances the outputs of the

cochleae to facilitate the processing of interaural level differences (ILDs; Darrow et al., 2006). However, counter to the latter suggestion, Larsen and Liberman (2010) have found that, in mice, a reduction in output from one cochlea (caused by damage) does not affect the output of the other cochlea.

More is known about the MOCR than the LOCR. This is largely because, unlike LOCR fibres, MOCR fibres are thick and myelinated (reviewed by Guinan et al., 1983), making direct recording and electrical stimulation easier (i.e. Hallin and Torebjork, 1973; Fitzgerald and Woolf, 1981). Moreover, MOCR effects can be measured non-invasively in humans using otoacoustic emissions (OAEs; e.g., Mountain, 1980; Siegel and Kim, 1982). OAEs can be evoked using a probe sound, which is typically a click or tone. When the probe enters the cochlea, some of its energy is reflected back out through the middle ear and into the ear canal, where it can be measured (van Dijk and Wit, 1990). This reflected energy is referred to as the OAE, and its amplitude depends upon the amount of cochlear amplification that is applied to the probe (Guinan, 1996). In classical OAE measurements of the MOCR, a noise, which is expected to elicit MOCR, is presented in the opposite ear to the probe. Presentation of such a noise “elicitor” has almost always been found to reduce the amplitude of the probe-evoked OAE (Collet et al., 1990; Veuille et al., 1991; Veuille et al., 1996; Backus and Guinan, 2006; Lilaonitkul and Guinan, 2009a). This effect is known as “OAE amplitude suppression”

and is widely believed to be due to the MOCR reducing the amount of cochlear gain applied to the probe (see Guinan, 1996, 2011).

However, as explained in Section 3.4, OAE amplitude is affected by many factors that are not related to cochlear gain, such as the degree and distribution of cochlear irregularities and the transmission properties of the middle ear. Changes in OAE amplitude therefore cannot be used to measure MOCR-induced cochlear-gain reduction quantitatively. Thus, because invasive methods cannot be used to measure MOCR effects in humans, the magnitude, and therefore functional importance, of the MOCR remains unknown. However, several psychophysical phenomena have been proposed to be the consequence of MOCR-induced cochlear-gain reduction; it may be possible to use these phenomena to measure the MOCR quantitatively (Strickland, 2001; Jennings et al., 2009; Aguilar et al., 2013; Yasin et al., 2014). The aim of this thesis is to establish which of these phenomena are associated with the MOCR and whether, or how, they can be used for quantitative measurements.

1.1 THE MEDIAL OLIVOCOCHLEAR REFLEX (MOCR)

The effect of the MOCR on the cochlear response to a probe sound changes as a function of the levels, frequencies, and timing of the probe and MOCR elicitor, and also depends on whether the elicitor is presented in the same or opposite ear to the probe. When searching for a psychophysical measure of MOCR-induced cochlear-gain reduction, it is

important to understand how the MOCR depends on these parameters. The following sections review the relevant literature regarding each parameter. Firstly though, the mechanisms thought to underlie the process of cochlear amplification, which the MOCR modulates, are described.

1.1.1 THE COCHLEAR AMPLIFIER

In the late 1940s, Thomas Gold argued that, because of the mammalian auditory system's remarkable sensitivity and ability to discriminate between sounds with very similar frequencies, there was likely to be an active amplification system within the cochlea (Gold, 1948). Since then, overwhelming evidence in support of this suggestion has emerged (see Ashmore et al., 2010 for review). Although the details of the mechanisms underlying cochlear amplification are still intensely debated, it is now well established that the OHCs play a crucial role (Davis, 1983; Brownell et al., 1985; Frank et al., 1999; Raphael et al., 2000; Recio and Rhode, 2000; Robles and Ruggero, 2001; Shera, 2001; Liberman et al., 2002; Breneman et al., 2009). Early evidence that the OHCs are involved in amplifying basilar membrane motion came from the observation that disabling the OHCs, using ototoxic drugs, caused behavioural threshold shifts and changes in neural tuning curves that were highly suggestive of a loss of cochlear amplification (Ryan and Dallos, 1975; Dallos and Harris, 1978; Liberman and Dodds, 1987). Since then, the discovery that the healthy cochlea spontaneously emits sound under certain conditions

(Kemp, 1978), which Gold (1948) predicted would be a consequence of active amplification, has confirmed that the cochlea is itself capable of independently generating sound energy.

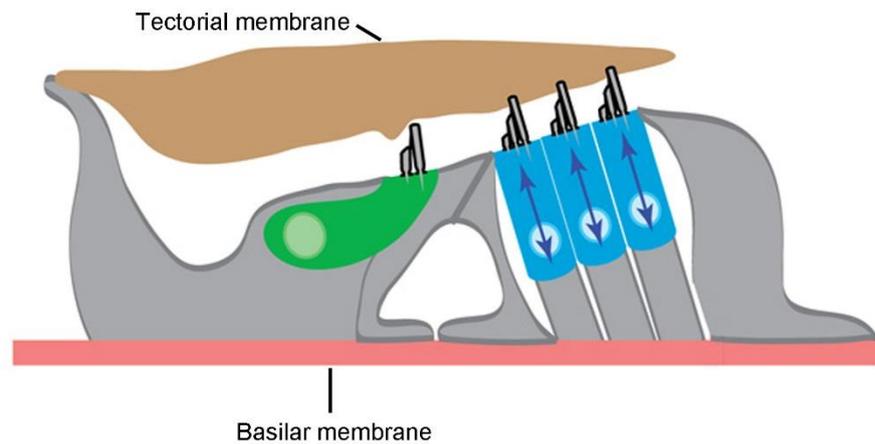


Figure 1.1: *Schematic representation of the cochlea. The inner and outer hair cells (IHCs and OHCs) are shown in green and blue respectively. The stereocilia extrude from the top of these cells towards the tectorial membrane. Cochlear amplification is thought to involve somatic motility (dark blue arrows) and/or motion of the OHC stereocilia. Image adapted from Peng and Ricci (2011).*

The OHCs sit parallel to the IHCs and are arranged in three to five rows, which run along the length of the basilar membrane. They consist of a cell body from which stereocilia extrude (see Figure 1.1). Two means by which the OHCs might provide amplification have been suggested. The first is by somatic motility allowing the cell body to contract and elongate in synchrony with the sound stimulus and thereby inject energy into the basilar membrane vibrations (see Figure 1.1; Dallos, 1992). The

contraction and elongation of the cell body is triggered by the opening of mechano-electrical transducer channels in the stereocilia (Chen et al., 2003; Verdon et al., 2003; Peng and Ricci, 2011). Voltage-dependent somatic motility was first demonstrated by Brownell et al. (1985) and such somatic electromotility has since been observed at frequencies that span the entire frequency range of mammalian hearing (Frank et al., 1999). There is substantial evidence that somatic motility results from activity of the motor protein prestin (Zheng et al., 2000). It has been shown that, with prestin genetically knocked out, cochlear sensitivity decreases (Liberman et al., 2002), supporting the suggestion that prestin, and therefore OHC somatic motility, is necessary for cochlear amplification. Further support for this idea comes from the observation that basilar membrane sensitivity and the sharpness of cochlear frequency tuning can be modulated by modulating the effectiveness of prestin (Santos-Sacchi et al., 2006).

The second proposed mechanism for cochlear amplification involves force generation by hair-bundle (stereocilia) motility (see Figure 1.1.). This is thought to be the amplification mechanism in non-mammalian vertebrates, which do not have OHCs (Hudspeth, 1997; Manley and Koppl, 1998). The mechanosensitive hair-bundles have been shown to produce oscillatory movements, or abrupt twitches, in response to force pulses on a sub-millisecond timescale, which can amplify basilar membrane motion (Crawford and Fettiplace, 1985; Martin and Hudspeth, 1999, 2001; Martin et al., 2001; Kennedy et al., 2005). Many

authors believe that both somatic motility and hair-bundle motility likely contribute to cochlear amplification in humans (see Dallos, 2008; Hudspeth, 2008; Ashmore et al., 2010).

1.1.2 MOCR-INDUCED COCHLEAR-GAIN REDUCTION AS A FUNCTION OF LEVEL

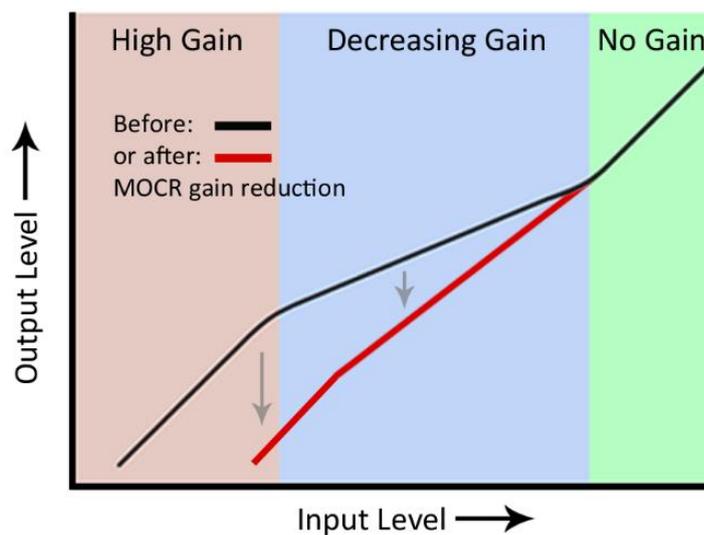


Figure 1.2: Schematic representation of the relationship between input sound level and the cochlear response size (output level). The relationship is shown both with (red) and without (black) MOCR-induced cochlear-gain reduction. Grey arrows highlight that the effect of MOCR-induced gain reduction is large at low levels and smaller at higher levels.

In 1971, Rhode demonstrated for the first time that, at the place that responds maximally to a sound stimulus, the size of the basilar membrane response does not grow linearly with increasing stimulus intensity; instead, it exhibits a compressive nonlinearity, growing at a

rate of < 1 dB/dB (Rhode, 1971). He also demonstrated that this nonlinearity disappeared after death, explaining why a similar observation had not been made in George von Békésy's earlier Nobel-Prize-winning work, where he measured the basilar membrane response to sound in cochleae from human cadavers (see von Békésy, 1960). It took more than a decade for Rhode's discoveries to be confirmed (Le Page and Johnstone, 1980; Sellick et al., 1982; Robles et al., 1986). Since that time, it has been shown that the size of the basilar membrane response as a function of stimulus level actually grows approximately linearly when the level is low (below ~ 30 dB SPL), and that it is only at medium levels (from ~ 30 dB SPL to ~ 80 dB SPL) that the response size grows compressively (Cooper and Rhode, 1992; Nuttall and Dolan, 1996; Ruggero et al., 1997). There is evidence that the cochlear response resumes linear growth at high stimulus levels (above ~ 80 dB SPL; Patuzzi et al., 1984; Ruggero and Rich, 1991; Ruggero et al., 1992; Rhode and Recio, 2000), although this remains somewhat controversial (Nuttall and Dolan, 1996; Ruggero et al., 1997; Cooper, 1998). One reason for this controversy is the difficulty in measuring the cochlear response to high level stimuli without damaging the cochlea (Robles and Ruggero, 2001). The black line in Figure 1.2 shows a schematised representation of the cochlear nonlinearity, with the stimulus intensity (input) plotted against the cochlear response size (output). There is a wealth of evidence from both psychophysical (Stelmachowicz et al., 1987; Oxenham and Plack, 1997; Nelson et al., 2001; Yasin et al., 2013b) and OAE measurements

(Kemp, 1978; Probst et al., 1991; Uppenkamp and Kollmeier, 1994; Veillet et al., 1996; Kalluri and Shera, 2007) that a similar nonlinearity exists in humans.

The cochlear nonlinearity arises because the amount of gain that the cochlear amplifier applies to a sound is dependent on the sound level (Dallos, 1992; Hudspeth, 2008). When the stimulus level is low, the gain is maximal and constant, and so the response grows at a constant, linear rate with increasing stimulus level (Figure 1.2: black line, portion of the function with the pink background). At medium levels, the amount of gain decreases progressively with increasing stimulus level, and so the cochlear response grows at a compressive rate (Figure 1.2: black line, portion of the function with the blue background). At high levels, it is thought that the cochlear response grows linearly because the cochlear amplifier applies no gain (Figure 1.2: black line, portion of the function with the green background).

When MOCR fibres are stimulated, cochlear amplification is reduced (Liberman et al., 1996; Cooper and Guinan, 2006). MOCR-induced gain reduction has the largest impact upon sounds that are subject to a large amount of cochlear gain to start with. It therefore affects low-level sounds more than higher-level sounds (Warren and Liberman, 1989a; Murugasu and Russell, 1996; Dolan et al., 1997; Cooper and Guinan, 2003, 2006; Guinan and Cooper, 2008). This is depicted in Figure 1.2 (red line), which shows that MOCR-induced cochlear-gain reduction makes

the cochlear input/output (IO) function more linear. The difference between the effect size at low and high input levels is highlighted by the grey arrows.

In order to elicit the MOCR, sounds need to be ~10 dB above hearing threshold. Above this level, the strength of the MOCR increases with increasing elicitor level (e.g., Warren and Liberman, 1989a; Collet et al., 1990; Ryan et al., 1991; Backus and Guinan, 2006). It is not known whether the MOCR effect eventually saturates at high elicitor levels, because these measures are confounded by activation of the middle-ear muscle reflex (MEMR), which, like the MOCR, reduces the size of the cochlear response.

The first MOCR effects were measured in the auditory nerve and were as large as 20-30 dB (Desmedt, 1962; Wiederhold and Kiang, 1970; Gifford and Guinan, 1987; Guinan and Gifford, 1988b). Quantitatively similar effects have been shown on IHC receptor potentials (Brown et al., 1983; Brown and Nuttall, 1984) and on basilar membrane motion (Murugasu and Russell, 1996, Dolan et al., 1997, Cooper and Guinan, 2003). In all of these studies, the size of the MOCR effect was quantified by calculating the stimulus level needed to produce the same response with and without electrical stimulation of the MOCR. Warren and Liberman (1989b) elicited the MOCR acoustically and found that, for an elicitor presented contralateral to the probe, MOCR effects on auditory nerve fibre firing rates were considerably smaller than those elicited by

electrical stimulation. The largest effect they measured was 12 dB, and the effect size varied dramatically with stimulus parameters, such as elicitor bandwidth and the temporal relationship between the elicitor and probe.

1.1.3 MOCR-INDUCED COCHLEAR-GAIN REDUCTION AS A

FUNCTION OF ELICITOR LATERALITY

MOCR effects can be evoked by elicitors that are presented either ipsilateral or contralateral to the probe (Liberman and Brown, 1986; Robertson et al., 1987; Brown, 1989). In many mammals with predominantly high-frequency hearing, such as mice, ~75% of MOCR fibres are “crossed” (project ipsilaterally) and ~25% are “uncrossed” (project contralaterally; Warr et al., 1986; Warr, 1992; Maison et al., 2003). This relationship is approximately reflected in the relative strengths of the ipsilateral and contralateral MOCR (Liberman and Brown, 1986; Gifford and Guinan, 1987; Brown, 1989; Maison et al., 2003). In humans, the ratio of crossed to uncrossed MOCR projections is unknown. However, in cats, chinchillas, and guinea pigs, which all have both low- and high-frequency hearing, the number of crossed and uncrossed MOCR fibres is more equal at lower, than at higher, frequencies (Iurato et al., 1978; Guinan et al., 1984; Robertson et al., 1987). In guinea pigs, Robertson and Gummer (1985) found that ~50% of all MOCR neurons measured responded best to ipsilateral acoustic stimulation, ~43% to contralateral stimulation, and ~7% responded

equally well to ipsilateral or contralateral stimulation. In new-world squirrel monkeys (Thompson and Thompson, 1986) and in old-world patas monkeys (Bodian and Gucer, 1980) the number of crossed and uncrossed MOCR fibres has also been found to be approximately equal. It may therefore be speculated that humans, who have predominantly low-frequency hearing, have similar numbers of crossed and uncrossed MOCR fibres.

OAE data from humans shows that the relative strength of ipsilaterally- and contralaterally-elicited MOCR gain reduction depends on the elicitor bandwidth. This suggests that the ipsilateral and contralateral MOCR pathways integrate over different frequency ranges. Lilaonitkul and Guinan (2009a) measured OAEs amplitude suppression for elicitors with a range of bandwidths (all presented at 60 dB SPL). The elicitor was presented either ipsilateral or contralateral to a 40-dB SPL probe tone, which was at either 0.5, 1, or 4 kHz. When the elicitor was narrowband, suppression by the ipsilateral elicitor was up to twice as strong as suppression by the contralateral elicitor. In contrast, ipsilateral and contralateral elicitor effects were similar when the elicitor was broadband. Noise elicitors presented bilaterally have been shown to produce the largest MOCR effects (Lilaonitkul and Guinan, 2009a, 2012), which would be expected, because bilateral elicitors activate both ipsilateral and contralateral MOCR pathways.

1.1.4 FREQUENCY DEPENDENCE AND TUNING OF MOCR-INDUCED COCHLEAR-GAIN REDUCTION

Physiological studies have shown that, when a high-frequency pure tone is played which maximally excited the base of the cochlea, the cochlear amplifier will amplify the travelling wave only at the place along the basilar membrane that is tuned to the frequency of the pure tone (e.g., Robles and Ruggero, 2001). Consequently, if the tone is subject to strong amplification, the basilar membrane response will be 'sharply tuned'; that is, the response will be large at the place tuned to the tone frequency and much smaller at places tuned to neighbouring frequencies. Tuning becomes broader when cochlear gain is reduced, as occurs when sounds are at a high level, when there is a hearing loss (Moore, 2007), or when the MOCR is activated (Wiederhold, 1970; Guinan and Gifford, 1988a; Cooper and Guinan, 2006). Less is known about the properties of amplification in the apex, because measurements from this part of the cochlea are much more difficult to make without causing damage to the cochlea which makes the results unreliable. However, there is evidence that, in the apex, amplification may not be applied in such a frequency-specific manner as in the base (e.g. Rhode and Cooper, 1996).

MOCR neurons usually have a distinct frequency to which they respond best (Guinan, 1996). In cats and guinea pigs, anatomical tracing of MOCR fibres suggests that each fibre projects approximately to the area of the cochlea that is tuned to the fibre's best frequency (Cody and Johnstone,

1982; Robertson, 1984; Robertson and Gummer, 1985; Liberman and Brown, 1986; Brown, 1989). It would therefore be expected that MOCR-induced cochlear-gain reduction is, at least to some extent, frequency-specific. Using OAEs in humans, Lilaonitkul and Guinan (2009a, b, 2012) and Maison et al. (2000) both found that MOCR effects are small when the elicitor bandwidth is narrow, and increase as the elicitor bandwidth increases up to 4-6.7 octaves. This suggests that the MOCR integrates information from almost the entire frequency-range of human hearing.

Lilaonitkul and Guinan (2009a, b, 2012) measured MOCR effects at different probe frequencies. They presented a narrowband-noise elicitor contralateral to a probe tone at either 0.5, 1, or 4 kHz, and found that the range of frequencies over which the MOCR could be elicited was dependent upon probe frequency. For the 0.5-kHz probe, tuning was broad with a skew towards higher frequencies; for the 1-kHz probe, tuning was broad, but with a skew towards lower frequencies (the most effective elicitor was 0.5-1 octave below the probe frequency in this case); and for the 4-kHz probe, tuning was narrow and centred on the probe frequency, but there was a broad low-frequency region in which the MOCR could also be elicited. The tuning properties found in these studies are in line with those found in cats (Warren and Liberman, 1989b), when it is taken into account that the range of hearing in cats is 1-1.5 octaves higher than in humans. Lilaonitkul and Guinan (2012) found that, in humans, for the 0.5-, 1- and 4-kHz probes, tuning was similar whether the elicitor was presented ipsilateral or contralateral to

the probe, although, for the 4-kHz probe, there was not a low-frequency region in which an ipsilateral elicitor could activate the MOCR. These findings are in agreement with studies in cats and guinea pigs, which also found little difference in tuning between the ipsilateral and contralateral reflex pathways (Cody and Johnstone, 1982; Robertson, 1984; Liberman and Brown, 1986; Brown, 1989).

As well as finding different tuning properties at different frequencies, Lilaonitkul and Guinan (2009a, b, 2012) found different absolute MOCR effect sizes at different frequencies. They found that MOCR effects became smaller towards frequencies in the middle of the frequency range of human hearing. This conflicts with findings in non-human mammals: in mice, the number of MOCR terminals per OHC increases around the central part of the cochlea (Maison et al., 2003) and in cats and guinea pigs, efferent effects are largest for MOCR fibres with medium or high best frequencies (Wiederhold, 1970; Teas et al., 1972; Guinan and Gifford, 1988a; Patuzzi and Rajan, 1990; Liberman, 1991). However, this apparent difference may merely reflect an OAE measurement artefact resulting from the fact that, in humans, OAE amplitudes are largest around 1 kHz and become smaller around the middle and higher part of the range of human hearing.

1.1.5 TIME COURSE OF MOCR-INDUCED COCHLEAR-GAIN

REDUCTION

Two distinct MOCR effects have been identified, distinguished by their slow and fast time courses. MOCR “fast effects” occur ~20-100 ms after the elicitor onset (Cooper and Guinan, 2003). Physiological measurements have shown that, in response to tones or noise bursts, the majority of MOCR efferents have response latencies of at least 10 ms; however, a few have latencies as short as 5 ms (Robertson and Gummer, 1985; Liberman and Brown, 1986; Brown, 1989; Brown et al., 2003). OAE estimates of the time course of MOCR-induced cochlear-gain reduction are in broad agreement with these findings: Backus and Guinan (2006) found latencies between the onset of the elicitor and the onset of MOCR effects on the probe OAE of ~25 ms, and James et al. (2002b) found a latency of 31-93 ms. The MOCR fast effect builds up to full strength ~100 ms after the elicitor onset and takes a similar time to decay after elicitation has ceased (Galambos, 1956; Desmedt, 1962; Fex, 1967; Wiederhold and Kiang, 1970; Gifford and Guinan, 1987; Backus and Guinan, 2006). Backus and Guinan (2006) found that the time course of the MOCR fast effect did not depend on the elicitor level (although they did not use a large range of elicitor levels) or whether the elicitor was presented ipsilateral or contralateral to the probe.

In animals, MOCR “slow effects” have an onset and offset time of 50-150 s (Cooper and Guinan, 2003) and, typically, reach their peak after

~120 s (Sridhar et al., 1995; Cooper and Guinan, 2003). Van Zyl et al. (2009) studied the effect of sustained MOCR stimulation in humans by measuring OAE amplitude suppression. They used a 16 minute long noise elicitor (interrupted twice for measurements) and found a sustained OAE amplitude suppression that was absent 1 minute after the elicitor was terminated. They found no evidence of a build-up of OAE amplitude suppression after the first minute of elicitor presentation. This suggests that the slow MOCR effect in humans is slightly faster than the effect found in animals, although it should be noted that few experiments have been performed in humans which address this subject.

The fast and slow MOCR effects are thought to be due to separate mechanisms: the fast effect is thought to be produced by MOCR-induced changes in OHC conductance, and the slow effect by changes in the stiffness of the OHCs (Sridhar et al., 1995; Dallos et al., 1997; Cooper and Guinan, 2003). However, the precise mechanisms by which cochlear amplification is reduced by the MOCR and indeed, as discussed earlier, the precise mechanisms by which amplification is produced by the OHCs, are still the source of much debate (Dallos, 2008; Hudspeth, 2008).

1.2 THE FUNCTION OF THE MOCR

Several possible roles for the MOCR in hearing have been suggested and these are reviewed below.

1.2.1 PROTECTION

The early development of the auditory system can be adversely affected by long-term noise exposure (Tallal et al., 1998; Chang and Merzenich, 2003; Sanes and Bao, 2009). Lauer and May (2011) found that mice whose MOCR had been disabled and who were reared in a noisy environment, showed evidence of auditory processing deficits that were not present in normal-hearing controls. This suggests that, by reducing the response of the auditory nerve to environmental noise, the MOCR helps to protect against the negative effects of long-term noise exposure during early development. There is also a range of evidence suggesting that the MOCR continues to protect the auditory system even after the early stages of development (reviewed by Rajan, 2000). In one study, anaesthetised guinea pigs were exposed to intense (105 dB SPL) tones at 6, 8, or 10 kHz. Exposure to such loud sound can cause a temporary reduction in hearing sensitivity, known as a “temporary threshold shift” (Reiter and Liberman, 1995). It was found that, for the higher frequency tones (8 or 10 kHz) and for the shorter exposure durations (1-2 minutes), the temporary threshold shifts were less severe when the olivocochlear bundle (OCB) was electrically stimulated during the

exposure. However, no such protective effect was observed for the 6-kHz tone or for longer exposure durations. It is thought that it is the slow, rather than the fast, MOCR effect that provides this protection (Reiter and Liberman, 1995; Cooper and Guinan, 2006). In another study in awake guinea pigs, animals were grouped depending on the strength of their MOCR, which was assessed using OAE suppression (Maison and Liberman, 2000). The guinea pigs were then exposed to an intense (109 dB SPL) noise stimulus for 4 hours. One week after exposure, auditory nerve compound action potentials were measured to assess the damage caused by the noise. It was found that animals with a weak MOCR suffered more damage than those with a strong MOCR. Liberman and Guinan (1998) have argued that the MOCR works in complement to the MEMR, with the MOCR providing protection at higher frequencies and the MEMR providing protection at lower frequencies.

Although numerous studies have found evidence that the MOCR is involved in protecting the auditory system from noise damage, it should be noted that some studies have found no evidence of a protective effect (Trahiotis and Elliott, 1970; Liberman, 1991). However, these are often not in direct conflict with the findings of the studies reviewed above. For example, Liberman (1991) found that transection of the OCB in cats had no effect on the severity of acoustic injury caused by exposure to an intense (100 dB SPL) pure tone at 6 kHz. This is in line with Reiter and Liberman's finding (discussed above) that the MOCR only provides protection at frequencies above 6 kHz.

1.2.2 SPATIAL HEARING

The medial SOC, from which the efferent MOCR fibres project, is thought to be the first site along the auditory pathway to encode interaural time difference (ITD) cues. The idea that the medial SOC may be involved in ITD processing arose both because it receives bilateral input from monaural brain stem nuclei, and because recordings of spike rate responses in the medial SOC have shown tuning to ITDs (Goldberg and Brown, 1969; Yin and Chan, 1990). In cats, Yin and Chan (1990) found that ~80% of cells in the medial SOC were sensitive to ITDs and Goldberg and Brown (1969) made a similar finding in dogs. This suggests that the MOCR might be involved in ITD processing. More recently, evidence has also emerged that suggests that the MOCR may play a role in ITD processing in humans; Francis and Guinan (2010) presented data showing that a noise elicitor can reduce the delay between the presentation of a probe and the arrival of the OAE in the ear canal (the OAE latency) by up to 0.5 ms. These latency changes are consistent with the MOCR reducing cochlear gain and broadening cochlear tuning (Oppenheim and Wilsky, 1997). Human binaural localisation in the horizontal plane is sensitive to ITDs of the order of 10 μ s (0.01 ms), and so these changes in cochlear response latency could have a profound effect on sound localisation. It is not yet known whether a mechanism exists to compensate for these delays, or whether they somehow assist sound localisation. The idea that the MOCR may assist sound localisation is supported by studies showing that accurate sound localisation is

dependent on normal MOCR function. For example, Fisch (1970) found a major reduction in the ability of humans to accurately localise sounds after unilateral transection of the OCB and May et al. (2004) found a reduction in the ability of cats to localise sounds after OCB lesions. An anatomical route through which a compensatory mechanism could operate has also been identified. As well as sending projections from the medial SOC to the cochlea, MOCR neurons send collaterals to and receives inputs from the cochlea nucleus (CN), which is before the SOC in the auditory processing hierarchy and is innervated by auditory nerve fibres (Thompson and Thompson, 1986; Robertson and Winter, 1988). It has been proposed that these collaterals send information about MOCR processing to the CN so that MOCR effects can be compensated for (Benson and Brown, 1990; Ye et al., 2000).

As well as involvement in ITD processing, it has also been suggested that the MOCR may help increase sensitivity to spatial cues in noisy environments. In support of this suggestion, a study in humans has found that sound localisation performance in listeners with a stronger MOCR (assessed by measuring OAE suppression) is less impaired by the presence of noise than in those with a weaker MOCR (Andeol et al., 2011).

1.2.3 ENHANCING DETECTION OF SIGNALS IN NOISE

It is often argued that the MOCR is involved in enhancing the audibility of sounds, like speech, in noisy environments (Giraud et al., 1997;

Liberman and Guinan, 1998; Clark et al., 2012). Numerous studies have found that MOCR stimulation can increase neural responses to brief sounds in low-level background noise (Nieder and Nieder, 1970; Winslow and Sachs, 1987; Dolan and Nuttall, 1988; Kawase et al., 1993). For example, in cats, Kawase and Liberman (1993) found that cutting the OCB led to a change in the auditory-nerve firing rate in response to a tone in noise that was equivalent to a ~6-dB decrease in signal-to-noise ratio; also in cats, Heinz et al. (1998) found that cutting the OCB efferents reduced vowel formant discrimination, measured behaviourally; and, in guinea pigs, Seluakumaran et al. (2008) found that electrical stimulation of the MOCR increased detection and frequency discrimination of tones in noise in neurons in the inferior colliculus. In a study by Dewson (1967), the ability of monkeys trained to detect human speech in noise was reduced after OCB transection, although it has since been suggested that this study may have been confounded because stapedius motor axons, which mediate the MEMR, may also have been cut (Kawase and Liberman, 1993). The evidence regarding the role of the MOCR in enhancing detection of signals in noise is not unequivocal, however; some studies have found no change in the discrimination of sounds in noise after OCB transection (Igarashi et al., 1972) or after functional MOCR lesions (May et al., 2002).

In humans, results have also been somewhat variable. Some patients showed a reduced ability to detect speech in noise after sectioning of the vestibular nerve (the nerve through which MOCR fibres exit the brain),

but others did not (Zeng and Shannon, 1994; Giraud et al., 1997; Zeng et al., 2000). It has been argued that sectioning of the vestibular nerve does not always cut the OCB (Chays et al., 2003), which may explain the inconsistency between these studies. There have also been several studies looking at whether there is a correlation between MOCR strength and psychophysical measurements of signal-in-noise detection in the same individuals. Some of these studies have found a positive correlation (e.g., Micheyl and Collet, 1996; Giraud et al., 1997; Micheyl et al., 1997; de Boer and Thornton, 2008). For example, Micheyl and Collet (1996) found a positive correlation between OAE amplitude suppression by a contralateral noise and improvements in the detection of a masked tone when a similar contralateral noise was presented. However, such effects are often small and variable, with some studies finding no correlation and others even finding a negative correlation (e.g., Mukari and Mamat, 2008; Wagner et al., 2008; de Boer et al., 2012). It has been argued that much of this variability may arise because a positive correlation would only be expected to occur in conditions where MOCR-induced cochlear-gain reduction affects the masker more than the signal (see de Boer et al., 2012). Collectively, these diverse findings demonstrate that, if the MOCR is involved in unmasking signals in noise, its role has not yet been well characterised. They also show that measurements of MOCR unmasking are very sensitive to stimulus parameters and suggest that there is a large degree of variability between subjects.

That the MOCR should be able to reduce masking is somewhat unintuitive given that, when the MOCR reduces cochlear gain, it will also reduce cochlear frequency selectivity. It might therefore be expected that, as in hearing impairment, MOCR-induced reduction in cochlear gain will *increase* masking by allowing maskers at more remote frequencies to become more effective. A recent modelling study has shown that, despite the reduction in frequency selectivity, significant unmasking is still possible under the assumption that the MOCR produces a gain reduction of at least 15 dB (Jennings et al., 2011; Chintanpalli et al., 2012).

There are two main ways in which the MOCR is thought to enhance signal detection in noise. The first is by reducing excitatory masking, whereby the signal response is overwhelmed by the masker response. It has been argued that, when the masker is at a lower level than the signal, an MOCR-induced reduction in cochlear gain will affect the masker more than the signal, and that therefore excitatory masking will be reduced (see Section 1.1.2; Winslow and Sachs, 1987; Kawase et al., 1993; von Klitzing and Kohlrausch, 1994; Guinan, 1996; Strickland, 2001; see Guinan, 2006). The second way in which the MOCR may enhance detection of a signal in noise is by reducing suppressive masking of the signal. Suppressive masking refers to a reduction in the amount of cochlear gain applied to the signal caused by the presence of the masker. Suppression is thought to be caused by the masker “jamming” the active process by which the signal’s response is amplified, although this

account has been disputed (see Section 2.4). The effectiveness of this jamming is assumed to be determined by the size of the masker response just basal to the peak response to the signal on the basilar membrane, because this is where the active process is thought to be located (see Patuzzi, 1996). Because the basilar membrane is tuned, with higher frequency sounds eliciting a peak response more basally than lower frequency sounds, this means that the masker will be most effective at suppressing the signal when its frequency is just above the signal frequency; in that case, the jamming will be produced by the masker peak response. It has been argued that, because the masker peak response is subject to cochlear gain, a reduction in gain as a result of MOCR activation would be expected to reduce the suppressive masking effectiveness of a masker with a frequency above the signal frequency (e.g., Viemeister and Bacon, 1982; Guinan, 1996; Wright, 1996; Strickland, 2004; this argument is laid out in full in Sections 2.1 and 2.3). It is this idea that the MOCR can produce a reduction in suppressive masking that is the focus of Chapter 2.

It has been argued that the MOCR may be under attentional control and that it may enhance listening in noise by frequency-selective control of cochlear gain (Giraud et al., 1995; Maison et al., 2001; de Boer and Thornton, 2007; de Boer et al., 2012). One way in which the MOCR might enhance signal detection in noise is by selective release from MOCR-induced cochlear-gain reduction in attended, compared to unattended, channels (Giard et al., 1994). Support for this hypothesis has been

provided by de Boer and Thornton (2007), who measured OAE suppression under conditions where the probe was either attended or unattended and found significantly less suppression when the probe was attended. This suggested that the gain of an attended stimulus is less reduced by the MOCR than the gain of an unattended stimulus. Conversely, Maison et al. (2001) found that attended stimuli are more efficient at eliciting the MOCR. They measured OAE suppression for a probe at either 1 or 2 kHz, whilst participants attended to either of these frequencies within the contralateral elicitor. They found greater OAE suppression at the attended than at the unattended frequency.

Further support for the idea that high-level control of the MOCR plays a role in signal detection in noise comes from studies showing a link between auditory learning and MOCR strength. In a training study, participants with the weakest OAE amplitude suppression before training showed the greatest improvement in a speech-in-noise task, and these participants also showed the largest increases in OAE amplitude suppression after training (de Boer and Thornton, 2008). In another study, children who had undergone an intensive programme of auditory training to help with problems in understanding speech in noise also showed an increase in OAE amplitude suppression (VeUILlet et al., 2007). Further evidence that the MOCR is under attentional control comes from studies showing that attentional state can modulate the response of the peripheral auditory system (Lukas, 1981; Froehlich et al., 1990; Avan and Bonfils, 1992; Froehlich et al., 1993; Harkrider and Bowers, 2009).

In one study using OAEs, Froehlich et al. (1993) found that performing an auditory task while OAE measurements were taken decreased OAE amplitudes. In another study, Harkrider and Bowers (2009) found that OAE amplitude suppression decreased (compared with passive listening) when listeners paid attention to either the ipsilateral probe or the contralateral noise elicitor. There is also evidence of cross-modal attention effects on peripheral auditory processing. For example, Puel et al. (1988), showed that a visual selective-attention task can have a significant impact on OAE amplitude. Visual attention has also been shown to modulate the size of cochlear responses in cats (Oatman, 1971, 1976) and chinchillas (Delano et al., 2007). Such high-level control of the MOCR is also anatomically plausible: in the rat, guinea pig, and gerbil, the SOC (from which the MOCR fibres originate) receives projections from both primary and non-primary auditory cortical areas (Budinger et al., 2000; Doucet et al., 2002; Coomes and Schofield, 2004). Although there is substantial evidence linking the MOCR to higher level processing, the ways and extent to which the MOC is under high-level control remains largely unknown.

1.3 A PUTATIVE PSYCHOPHYSICAL CONSEQUENCE OF THE MOCR

It has been argued that the putative unmasking function of the MOCR might manifest, under laboratory conditions, in the so-called “overshoot” or “temporal” effect (von Klitzing and Kohlrausch, 1994). Here, overshoot refers to the fact that a signal presented at the onset of a masker is easier to detect when the masker is preceded by a “precursor” sound (Zwicker, 1965a). There is evidence both in support of and against this suggestion. In support, it has been observed that the increase in the magnitude of overshoot with increasing precursor duration is similar to the build-up time of the MOCR. For example, McFadden et al. (2010) and Walsh et al. (2010) both found no overshoot for noise precursors with a duration less than 20-30 ms, and a steady increase in the amount of overshoot with increasing precursor duration up to 150-200 ms. Walsh et al. (2010) found that the time course of this effect was similar to the time course of OAE amplitude suppression measured in the same participants; their MOCR time-course estimates conformed well with those made by other researchers (e.g., James et al., 2002a; Backus and Guinan, 2006). It has also been argued that overshoot is likely linked to the MOCR, or at least to processes within the cochlea, because overshoot is absent under conditions where cochlear amplification is lost (Strickland, 2001), such as permanent cochlear hearing-loss (Bacon and Takahashi, 1992), cochlear-based hearing loss caused by ingestion of

ototoxic drugs (McFadden and Champlin, 1990), or temporary threshold shift resulting from exposure to intense sound (Champlin and McFadden, 1989). However, this does not necessarily imply a cochlear origin because cochlear damage will have a major impact on post-cochlear processing.

Other studies have provided evidence against the idea that overshoot is related to the MOCR. For example, if the MOCR is involved in overshoot, then overshoot should still occur when the precursor is presented contralateral to the masker and signal, where it would still be expected to activate the MOCR (see Section 1.1.3). However, no such effect has been measured, either with a tonal (Bacon and Healy, 2000) or a noise (Bacon and Liu, 2000; Savel and Bacon, 2003) precursor. Furthermore, Kidd and Wright (1994) found that presenting a noise precursor diotically gave the same amount of overshoot as presenting the precursor only to the ear that contained the signal, although the diotically presented precursor would have been expected to be a more effective elicitor of the MOCR (see Section 1.1.3).

A salient feature of overshoot, which might give insight into the mechanisms that underlie it, is the importance of masker and precursor energy above the signal frequency (McFadden, 1989; Bacon and Smith, 1991; Schmidt and Zwicker, 1991; Fletcher et al., 2013). Bacon and Viemeister (1985) measured overshoot for a 1-kHz signal with a masker and precursor that were either below, at, or above the signal frequency.

They found that the largest overshoot was produced by maskers and precursors that were higher in frequency than the signal. In another study, Schmidt and Zwicker (1991) compared the amount of overshoot produced by a broadband noise, high-pass noise, and low-pass noise. They found a similar large overshoot with both the broadband and high-pass noise masker and precursor, but much less overshoot for the low-pass noise masker and precursor. The importance of masker and precursor energy above the signal frequency, and the fact that suppressive masking by a masker with a frequency above the signal frequency is thought to depend on the amount of cochlear gain applied to the masker (see Section 1.2.3), has led to the suggestion that overshoot is due to a reduction in suppressive masking by the precursor (e.g., Strickland, 2008). It has been argued that the precursor might elicit the MOCR, causing a reduction in the amount of cochlear gain applied to the masker and therefore a reduction in suppressive masking of the signal. This possibility is explored in Chapter 2.

1.4 PSYCHOPHYSICAL MEASUREMENTS OF COCHLEAR GAIN AND COMPRESSION

Over the last few decades, there has been significant progress in the development of methods for measuring cochlear gain and compression psychophysically. More recently, attempts have been made to measure the MOCR using these methods. This section reviews the development of the various methods.

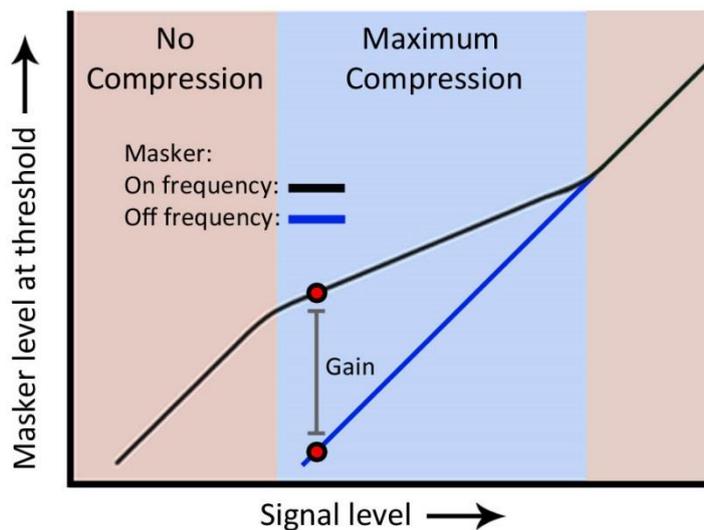


Figure 1.3: Schematic representation of the expected growth of signal maskability with a masker which is either at or below the signal frequency (“on-” and “off-frequency”, shown in black and blue, respectively), based on the method proposed by Stelmachowicz et al. (1987). The amount of gain applied to the on-frequency masker peak response is assumed to be equal to the difference in the masker level at threshold between the on- and off-frequency masker for a given signal level (shown by the grey line).

Stelmachowicz et al. (1987) argued that cochlear compression can be measured psychophysically by comparing the masker level needed to just mask a signal with a fixed level when the masker is either at or below the signal frequency ("on-" and "off-" frequency maskers). An idealised plot of the expected signal level as a function of the masker level at threshold [the "growth of maskability" (GOM)] for the on- and off-frequency maskers is shown in Figure 1.3. The masker level at threshold for the on-frequency masker is dependent on the amount of cochlear gain that the masker is subject to, but for the off-frequency masker, the threshold should be independent of gain (Schmiedt and Zwislocki, 1980; Weber, 1986; Robles and Ruggero, 2001). If it is assumed (i) that any gain applied to the signal affects the on- and off-frequency masker level at threshold equally, (ii) that the on-frequency masker masks the signal by its peak response and the off-frequency masker by its passive tail, and (iii) that the only difference between the masker peak and tail responses is the cochlear gain applied to the peak response (see, however, Chapter 3), then the difference between the on- and off-frequency masker level at threshold at a given signal level will be equal to the amount of cochlear gain applied to the on-frequency masker peak response (see Figure 1.3, grey line). Furthermore, because masking by an off-frequency masker is not subject to any cochlear gain, it is not subject to any cochlear compression. Therefore, under the assumption that any compression of the signal would affect the masker level at threshold for the on- and off-frequency maskers equally, the difference in GOM between the on- and

off-frequency masker would be expected to reflect the amount of cochlear compression associated with the on-frequency masker. Thus, in Figure 1.3, compression is only seen at medium levels, where the GOM for the on-frequency masker is slower than the GOM for the off-frequency masker.

Stelmachowicz et al. (1987) measured GOM for an on- and off-frequency masker in a simultaneous-masking paradigm and estimated a “compression ratio”, which is the ratio of the shallowest slopes of the GOM functions for the on- and off-frequency maskers. They found a compression ratio of $\sim 2:1$ for normal-hearing listeners, and a reduced compression ratio (close to $1:1$) in hearing-impaired listeners. This reduced compression is expected, because hearing impaired listeners typically have reduced cochlear gain (Evans, 1975; Evans and Harrison, 1976). However, using physiological measures of basilar membrane displacement in animals, a compression ratio of $\sim 6:1$ has been found (Murugasu and Russell, 1996; Ruggero et al., 1997; Russell and Nilsen, 1997), which is much greater than that measured in normal hearing listeners by Stelmachowicz et al. (1987). This discrepancy may have been due to suppressive masking of the signal by the off-frequency masker. Off-frequency maskers are potent suppressors (e.g., Moore and Vickers, 1997), and so suppression may have reduced the apparent difference in masking effectiveness between the on- and off-frequency masking conditions, causing the amount of gain and compression to be underestimated.

To avoid suppressive masking of the signal, Oxenham and Plack (1997) used a similar paradigm to Stelmachowicz et al. (1987), but with forward, rather than simultaneous, maskers. A forward masker would not be expected to suppress the signal, because suppression is thought to be instantaneous (Arthur et al., 1971). With forward masking, Oxenham and Plack (1997) found a compression ratio of ~5:1, which is more in line with the physiological estimates from animals.

Like Stelmachowicz et al. (1987), Oxenham and Plack (1997) measured the masker level needed to just mask the signal, with the signal level fixed. At medium and high signal levels, the basilar membrane response to the signal will spread basalwards (Robles and Ruggero, 2001), allowing listeners to detect the signal in frequency channels that are remote from the signal frequency ("off-frequency listening"; Patterson and Nimmo-Smith, 1980; O'Loughlin and Moore, 1981). These remote frequency channels are subject to less cochlear gain (Robles and Ruggero, 2001) and, therefore, off-frequency listening will lead the amount of cochlear gain to be underestimated. To limit off-frequency listening, Oxenham and Plack (1997) presented a high-pass or notched noise at a low level to mask remote frequency channels.

Nelson et al. (2001) proposed another solution. They made the signal more or less easy to mask by adjusting the temporal gap between the masker and the signal, rather than by adjusting the signal level, and measured the masker level at threshold as a function of masker-signal

gap. This so-called “temporal masking curve” (TMC) method allowed them to fix the signal level at a low value, where the basilar membrane response would not spread basalwards (Robles and Ruggero, 2001), and therefore to avoid off-frequency listening. The TMC method assumes that the post-cochlear masking effect at a given signal frequency decays in the same way across masker frequency. This assumption is supported by work showing that gap-detection thresholds are approximately constant across frequency, but may hold only for short maskers (less than ~ 30 ms; Shailer and Moore, 1987; Wojtczak and Oxenham, 2010). Using this method, Nelson et al. (2001) estimated a compression ratio which is similar to physiological findings.

Several studies have attempted to measure MOCR-induced cochlear gain change using the TMC method. Roverud and Strickland (2010) and Krull and Strickland (2008) measured TMCs for an off-frequency masker and a 4-kHz signal, with and without a tonal, ipsilateral MOCR-elicitor at the signal frequency. Based on their results, they estimated the elicitor caused a cochlear-gain reduction of up to 20 dB. However, they were unable to control for any direct, post-cochlear masking of the signal by the elicitor (discussed in Chapter 3, Sections 3.1 and 3.2.1). Aguilar et al. (2013) also measured TMCs for an off-frequency masker and 4-kHz signal with and without an elicitor present. In this case the elicitor was a broadband noise. Aguilar et al. (2013) addressed the issue of post-cochlear masking by presenting their noise elicitor contralateral to the signal and masker, where it produced little post-cochlear masking of the

signal. They found no MOCR-induced gain reduction by the elicitor. One possible reason for the absence of an MOCR effect is that they used a long masker (200 ms). The masker may itself have elicited the MOCR (see Section 1.1.4) and thus resulted in an underestimate of MOCR-induced cochlear-gain reduction by the elicitor. Yasin et al. (2014) used a different approach to control for post-cochlear masking by the elicitor. They, like Roverud and Strickland (2010) and Krull and Strickland (2008), used an ipsilateral elicitor, but increased the signal level in the with-elicitor conditions, so that the signal would be equally detectable in the with- and without-elicitor conditions. Yasin et al. (2014) measured cochlear gain and compression using a method related to the TMC method, known as the “fixed-duration masking curve” (FDMC; Yasin et al., 2013b, a). In the FDMC method, the signal is made more or less detectable by changing the relative duration of the signal and the masker, rather than by changing the masker-signal gap. Using this approach, they measured an elicitor effect of up to 25 dB, on average. However, because Yasin et al. used an ipsilateral elicitor, it is possible that the large elicitor effects observed in that study were caused by direct nonlinear interactions due to partial temporal overlap between the cochlear responses to the elicitor and masker. This possibility is explored in Chapter 4.

1.5 SUPPRESSION OF OTOTACOUSSTIC EMISSIONS CAUSED BY ELICITATION OF THE MOCR

As discussed earlier (Section 1.1.), the amplitude of an OAE evoked by a click or tone in one ear [referred to as “evoked” OAEs (EOAEs)] can be reduced by presenting a sound to the opposite ear (OAE amplitude suppression) and this is widely believed to be due to the MOCR reducing the amount of cochlear gain applied to the probe (see Guinan, 1996, 2011). There are two main reasons for this belief. Firstly, the MOCR pathway is the only known neural pathway that projects from one cochlea to the other and can affect the cochlea’s mechanical response, and thereby affect the OAE amplitude. Secondly, electrical or acoustic activation of the MOCR has been shown to produce effects on the cochlear or neural response to sounds that are qualitatively similar to OAE amplitude suppression effects (Warren and Liberman, 1989b, a; Guinan, 2011; Lilaonitkul and Guinan, 2012).

As well as being evoked using a single probe tone or click, OAEs are also commonly evoked using two tones at different frequencies (f_1 and f_2). The OAE that is measured is a distortion product resulting from the combination of these two probe tones, usually at $2 \cdot f_1 - f_2$, where $f_1 > f_2$ (Siegel et al., 1982). These OAEs are therefore known as distortion-product OAEs (DPOAEs). The interpretation of the effect of an MOCR elicitor on DPOAEs is often complex. This is because the change in DPOAE

amplitude is the result of changes to two components, which may each be affected differently by the MOCR (Siegel and Kim, 1982; Moulin et al., 1993a; Shera and Guinan, 1999; Abdala et al., 2009; Henin et al., 2011). If the MOCR reduces the amplitude of only one component, and the phase relationship between the two components is such that they cancel each other, then an MOCR-induced reduction in cochlear gain could actually cause an increase in the DPOAE amplitude (Guinan, 2011).

Despite the fact that OAE amplitude suppression, whether measured using EOAEs or DPOAEs, is not a quantitative measure of MOCR effects, OAEs remain an important tool for determining whether or not a sound has elicited the MOCR, and also offer a means to qualitatively compare the effect of different elicitors. OAEs may offer a valuable independent means of validating psychophysical measurements of MOCR effects. For that reason, OAE measurements are performed alongside psychophysical measurements in the experiments presented in Chapters 3 and 4. In these experiments, EOAEs are preferred to DPOAEs because the results are more straightforward to interpret.

1.6 THESIS OVERVIEW

The aim of the work reported in this thesis was to find a quantitative measure of the MOCR in humans using psychophysical measurements. The thesis is split into five chapters, including this introductory chapter. A brief summary of each of the remaining chapters follows.

The aim of the work reported in Chapter 2 was to establish whether overshoot arises as a result of MOCR activation. In particular, the chapter examines whether the precursor elicits an MOCR-induced cochlear-gain reduction which reduces suppressive masking of the signal (see Section 1.3). Overshoot was measured for a 4-kHz sinusoidal signal and a 4.75-kHz sinusoidal masker and precursor. In the same set of participants, a forward-masking paradigm was used to measure the amount of suppressive masking exerted by the masker, with and without the precursor present. While the precursor yielded strong overshoot, and the masker produced strong suppression, there was no evidence of any precursor-induced reduction in suppressive masking. Predictions based on an established model of the cochlear IO function indicate that the failure to measure any reduction in suppression when the precursor was present was unlikely to represent a false negative outcome. These findings suggest that overshoot is not a perceptual consequence of the MOCR. Overshoot was therefore not pursued as a potential quantitative measure of the MOCR in subsequent experiments.

The aim of the work reported in Chapter 3 was to develop a method for directly measuring MOCR effects on cochlear gain by further developing the TMC method for measuring cochlear-gain reduction by a contralateral elicitor (see Section 1.4). Unlike in a recent study by Aguilar et al. (2013), the experiment presented in Chapter 3 used a short masker that would not itself be expected to elicit an MOCR effect on the signal that could have led the elicitor effect to be underestimated (see Section 1.4). Moreover, in order to control for any direct, post-cochlear masking of the signal by the elicitor, TMCs were measured not just for an off-frequency masker, but also for an on-frequency masker (see Chapter 3, Section 3.2.1). A new method for estimating cochlear gain, cochlear compression, and elicitor-induced cochlear-gain reduction from TMC data was also developed, in which the entire dataset is fitted with a generic model of the cochlear IO function. The cochlear IO function model produced an excellent fit to both the with- and without-elicitor TMCs. The model estimates of cochlear gain and cochlear compression without the elicitor accorded well with previous psychophysical estimates in humans, as well as with physiological estimates from non-human animals. To verify that the elicitor had caused an MOCR-induced reduction in cochlear gain, OAE suppression was measured in the same participants using the same elicitor. OAE suppression results indicated that the elicitor caused a reduction in cochlear gain and the psychophysical results indicated that this gain reduction amounted to ~ 4 dB, on average.

The aim of the work reported in Chapter 4 was to assess the validity of recent findings by Yasin et al. (2014), who estimated an MOCR-induced cochlear-gain reduction caused by a low-level narrowband-noise elicitor that was four times larger than that found in Chapter 3. Unlike in the experiment reported in Chapter 3 of this thesis, Yasin et al. (2014) presented their elicitor ipsilaterally and controlled for post-cochlear masking by adjusting the signal level (see Section 1.4). To estimate cochlear-gain reduction, Yasin et al. (2014) used the FDMC method, which is similar to the TMC method used in Chapter 3 (see Section 1.4). The experiment reported in Chapter 4 used the FDMC method to estimate the amount of gain reduction caused by a long ipsilateral elicitor, like the one used by Yasin et al. (2014), and compared it to the amount of gain reduction caused by a much shorter ipsilateral elicitor. The short elicitor was presented at a level so that it would produce the same amount of masking as the long elicitor, but was too short to activate the MOCR in time to affect the signal detectability. The long and short elicitors both caused large psychophysical effects, indicating either that the MOCR acts more quickly than previously thought, or that the effect was not due to MOCR-induced cochlear-gain reduction. OAE suppression was also found for both the long and short elicitors. In Chapter 4, it is argued that both the OAE and psychophysical effects of the short and long elicitors may, at least in part, be the result of nonlinear interactions between the elicitor and the masker as a result of direct temporal overlap of their cochlear responses.

The final chapter summarises the findings of the thesis and discusses how they relate to auditory research more broadly. This chapter also highlights important questions that have been raised by the experimental results, and suggests several important areas for future research. Probably the most exciting possibility is that the improved approach to measuring cochlear gain and MOCR-induced cochlear-gain reduction developed in this thesis could become a valuable audiometric profiling tool, and may give insight into the individual differences that underlie hearing problems in audiometrically normal listeners. Parametric exploration of the MOCR using this approach may also allow the functional importance of the MOCR in humans to be better understood.

CHAPTER 2

IS OFF-FREQUENCY OVERSHOOT A CONSEQUENCE OF THE MEDIAL OLIVOCOCHLEAR REFLEX REDUCING SUPPRESSIVE MASKING?

2.1 INTRODUCTION

The ability to hear out a signal from a background sound can be improved by a preceding sound (henceforth referred to as precursor). As discussed in Section 1.3.1, one instance of this kind of context-dependent change in signal audibility is the so-called “overshoot” or “temporal” effect, whereby the detectability of a signal can be degraded when it is

presented at the onset of a masker, rather than after a delay (Zwicker, 1965a). Another instance of context-dependent change in signal audibility is “enhancement”, which refers to the phenomenon whereby a spectral region in a complex sound “pops out” (i.e., becomes more salient) when that region is preceded by its spectral complement (Schouten, 1940; Viemeister, 1980).

Overshoot is observed only when the signal is shorter than about 20 ms (Fastl, 1977). In contrast, enhancement persists even when the signal is hundreds milliseconds long (e.g., Summerfield et al., 1984; Carlyon, 1989; Thibodeau, 1991). Measurements of enhancement have used precursors with energy both above and below the signal frequency, but no, or reduced, energy at the signal frequency (e.g., Viemeister et al., 2013). In contrast, overshoot is typically measured using broadband precursors and maskers, with energy both at and away from the signal frequency. However, it has been shown that substantial overshoot is also observed when the precursor and masker are narrowband, but only when their frequency is sufficiently different from the signal frequency (Zwicker, 1965b; Bacon and Smith, 1991). With broadband precursors and maskers, it is thought that both the on- and off-frequency energy within them contribute to overshoot (henceforth referred to as “on-” and “off-frequency overshoot”), albeit by different mechanisms. The mechanism proposed to underlie off-frequency overshoot (Strickland, 2004, 2008) is similar to that proposed to underlie enhancement (Viemeister and Bacon, 1982): in both phenomena, the signal response

is thought to be increased in the presence of the precursor, because suppression of the signal by the masker is reduced, or “adapted”. In off-frequency overshoot, the suppression by the masker is thought to occur in the cochlea (referred to as two-tone suppression) and the adaptive effect of the precursor is thought to be mediated by the MOCR (Strickland, 2004). In enhancement, suppression and adaptation of suppression are thought to occur more centrally, possibly involving neural adaptation and lateral inhibition (Palmer et al., 1995; Wright, 1996; Nelson and Young, 2010). For enhancement, the adaptation-of-suppression hypothesis has been tested explicitly. In particular, it has been shown that an enhanced signal causes more forward masking than an unenhanced signal, indicating that it elicits a larger response (Viemeister and Bacon, 1982; Thibodeau, 1991; Byrne et al., 2011). For off-frequency overshoot, however, the adaptation-of suppression hypothesis has not yet been explicitly tested.

The aim of the current study was to conduct this test. The most off-frequency overshoot is produced when the masker (and precursor) frequency is higher than the signal frequency (Schmidt and Zwicker, 1991). Higher-frequency maskers also produce more suppression (referred to as high-side suppression) than lower-frequency maskers (low-side suppression; Shannon, 1976; Duifhuis, 1980; Cooper, 1996). Psychophysical and physiological studies have shown that low-side suppression grows roughly linearly with masker level, whereas high-side suppression grows compressively (Duifhuis, 1980; Javel et al., 1983;

Costalupes et al., 1987; Delgutte, 1990; Cooper, 1996; Yasin and Plack, 2007). This suggests that low-side suppression is caused by the tail, and high-side suppression by the peak, of the masker's travelling wave response (see Patuzzi, 1996 for a detailed discussion of this hypothesis). The peak amplitude of the travelling-wave response depends on the amount of cochlear amplification (Robles and Ruggero, 2001). Thus, when the masker frequency is higher than the signal frequency, a reduction in the masker amplification through activation of the MOCR should reduce the amount of suppression caused by the masker, which would, in turn, increase the response to the signal.

In the current study, overshoot was measured for a short sinusoidal signal at 4 kHz, with a sinusoidal masker and precursor at 4.75 kHz (Figure 2.1A). To maximize the chances of finding overshoot in all individuals, the precursor and masker were presented continuously, without a gap. This means that at least some part of the measured overshoot may have been caused by a reduction in central masking effects, such as transient masking (Bacon and Moore, 1987) or diversion of attention (Scharf et al., 2008) by the masker onset: Without the precursor, the masker onset could be confused with the signal onset or draw attention away from the signal frequency towards the masker frequency. Continuous presentation of the precursor and masker removes the masker onset and thus eliminates these effects, making the signal more clearly audible. In order to test whether any part of the measured overshoot was caused by adaptation of suppression, in this

study, the suppression of the signal by the masker was measured both with and without the precursor present. For that, the forward-masking effectiveness of the signal alone and the signal and masker combined were measured first. A reduction in the forward-masking effectiveness of the signal by the masker would be assumed to be indicative of suppression (see, for example, Houtgast, 1972; Shannon, 1976). Importantly, the signal and masker durations were chosen so that the signal and masker would not have been able to elicit the MOCR in time to influence the amount of forward masking (12.5 ms; see Wojtczak and Oxenham, 2010). Then, the forward-masking effectiveness of the signal when presented together with the masker and the precursor was measured. A reduction in suppression due to the precursor (adaptation of suppression) would be expected to increase the forward-masking effectiveness of the signal.

In principle, the approach taken in this study is similar to that used by Viemeister and Bacon (1982) for enhancement. There are, however, crucial differences. Firstly, their precursor and masker were broadband with a spectral notch, whereas those used here were sinusoidal. Secondly, their signal and masker were almost ten times longer than those used here (100 versus 12.5 ms). Other studies on enhancement have used even longer signals and maskers (Thibodeau, 1991). Whereas Viemeister and Bacon found evidence for adaptation of suppression in enhancement, no such evidence was found here in overshoot, despite the finding of substantial overshoot and suppression. Predictions based on

an established model of the cochlear IO function indicate that the failure of the present study to find adaptation of suppression in overshoot is unlikely to represent a false negative outcome.

2.2 METHODS

2.2.1 GENERAL OUTLINE

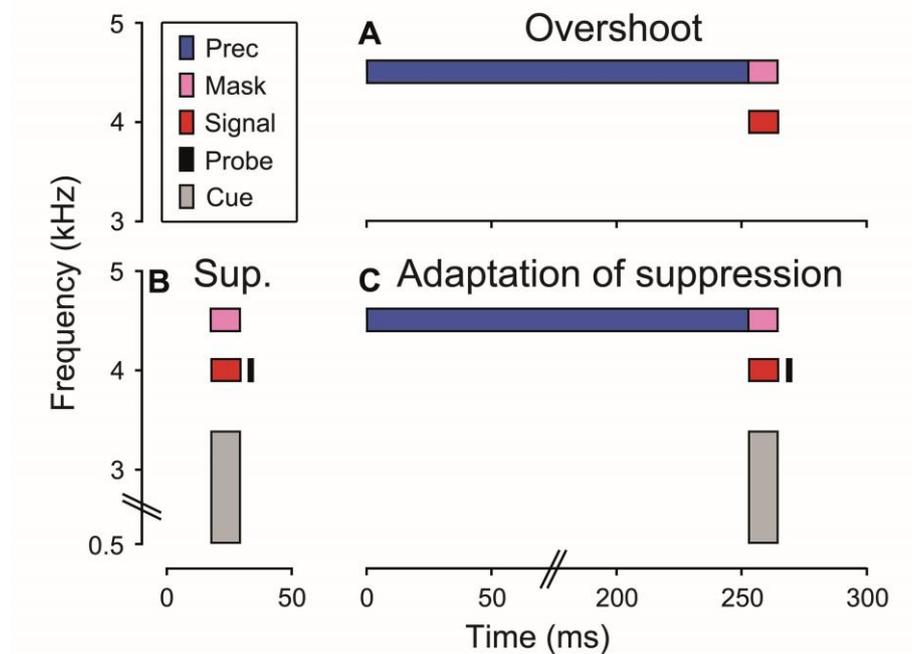


Figure 2.1: Schematic representation of the spectral and temporal characteristics of the stimuli used in the overshoot (A), suppression (B) and adaptation of suppression (C) experiments. The different stimuli (signal, probe, masker and precursor) are represented by different colours (see legend in panel A). The overshoot experiment used a 2.5- and 12.5-ms signal duration; only the 12.5-ms duration is shown here. The precursor duration is not to scale.

This study consisted of three experiments. In the first experiment, referred to as the “overshoot experiment” (Figure 2.1A), overshoot was measured for a 4-kHz sinusoidal signal and a sinusoidal masker and precursor at one auditory-filter bandwidth (equivalent rectangular bandwidth (ERB); Glasberg and Moore, 1990) above the signal frequency (4.75 kHz). The precursor and masker had durations of 252.5 and 12.5 ms, respectively, and were presented continuously and at the same level. The signal had a duration of 2.5 ms, initially, and was gated on together with the masker. The 2.5-ms duration was used, because shorter signal durations have yielded larger overshoot effects in previous studies (Zwicker, 1965b). Subsequently, overshoot was remeasured with a 12.5-ms signal duration (referred to as “supplementary overshoot experiment”), because this was the signal duration used in the other two experiments. In this case, the signal and masker were gated on and off together. In both overshoot experiments, the signal detection threshold was measured both with and without the precursor present. Overshoot is the difference in signal detection threshold between these two conditions.

Given that, in the overshoot experiment, the masker frequency was higher than the signal frequency, the masking effect would be presumed to have been predominantly suppressive (e.g., Duifhuis, 1980; Cooper, 1996). The second experiment, referred to as the “suppression experiment”, used a forward-masking paradigm to quantify the amount of suppression exerted by the masker (Figure 2.1B). In the suppression

experiment, the signal duration was 12.5 ms and the detection threshold was measured for a sinusoidal probe stimulus, presented 2.5 ms after the signal offset. The probe had the same frequency as the signal and a duration of 2.5 ms. The probe detection threshold was measured in the presence of either the signal alone or the signal and masker combined. The signal and masker were gated on and off together. Any suppression exerted by the masker would decrease the size of the signal response, which, in turn, would decrease the probe detection threshold (i.e., make the probe easier to detect). The signal and masker levels were set individually for each participant. First, the signal detection threshold was measured in quiet and the signal level was set to 25 dB sensation level (SL). Then, the masker level was set so that the masker would just render the 25-dB SL signal inaudible. In the overshoot experiment, the masker and precursor were set to the same level as in the suppression experiment. Stimuli of similar durations and levels as in the suppression experiment have been used previously to measure cochlear compression using forward masking (Yasin et al., 2013b).

According to the adaptation-of-suppression hypothesis, the precursor would be expected to reduce the amount of suppression exerted by the masker in the overshoot experiment. The aim of the third experiment, referred to as the “adaptation-of-suppression experiment”, was to quantify any precursor-induced reduction in suppression using the same forward-masking paradigm as used in the suppression experiment. Any precursor-induced reduction in suppression would increase the signal

response and thus manifest as an increase in the probe detection threshold.

Control measurements were conducted to measure the probe detection threshold in the presence of the masker alone and the masker and precursor combined. This was to assess the direct masking effects of the masker and/or precursor on the probe. The timing of the stimuli was the same as in the suppression and adaptation-of-suppression experiments.

2.2.2 STIMULI

All stimuli were gated on and off with 2.5-ms quarter-sine and -cosine ramps, respectively $[\sin\left(\frac{\pi}{2} \cdot \frac{t}{2.5}\right)$ and $\cos\left(\frac{\pi}{2} \cdot \frac{t}{2.5}\right)$, where t is time in ms].

All stated durations of stimuli, and gaps between stimuli, refer to the time between the 3 dB-down (half-power) points of the stimulus ramps. The phases of the masker, signal and probe were randomised between trials. The phase of the precursor was set such that there was no phase discontinuity between the precursor offset and the masker onset.

In the suppression and adaptation-of-suppression experiments, a cue was presented to disambiguate the signal from the probe. The cue was gated on and off simultaneously with the signal. It was a 15-ERB wide noise, centred 9 ERBs below the signal frequency. It was filtered so as to produce equal excitation per ERB within its passband (Glasberg and Moore, 2000) and presented at 30 dB SPL/ERB. It was confirmed that, at this level, the cue did not produce any significant masking of the probe.

When measuring the masker level needed to render the signal inaudible, it was difficult to hear out the signal when the masker and signal were gated on and off simultaneously. Therefore, in these measurements, the masker duration was increased so that the masker onset preceded the signal onset by 10 ms. The offsets remained simultaneous.

All stimuli were generated digitally at a sampling rate of 24.414 kHz using TDT System 3 (Tucker-Davies Technologies, Alachua, FL, USA) and MATLAB (The MathWorks, Natick, MA, USA). They were digital-to-analogue converted with a 24-bit amplitude resolution (TDT RP2), amplified (TDT HB7), and presented monaurally to the left ear using Sennheiser HD 600 headphones (Wedemark-Wennebostel, Germany). Participants were seated in a double-walled, sound-attenuating booth (IAC, Winchester, UK).

2.2.3 EXPERIMENTAL PROTOCOL

The study was conducted in four consecutive stages. Firstly, participants were screened for normality of hearing. Secondly, detection thresholds in quiet were measured for the probe and 12.5-ms signal used in the suppression and adaptation-of-suppression experiments. For the probe, the detection threshold was also measured in the presence of the cue stimulus (see Section 2.2.2). Thirdly, the masker level needed to just render inaudible the 12.5-ms signal at 25 dB SL was measured. Finally, the conditions from the overshoot, suppression and adaptation-of-

suppression experiments, as well as the control experiment, were measured in a random order. The supplementary overshoot experiment was measured after the other conditions, with a partially different set of participants.

Overall, the study lasted around 13 h, depending on the amount of training needed for performance to stabilise. The study was conducted over several days and included regular breaks

2.2.4 PROCEDURE

All thresholds were measured using a three-interval, three-alternative forced-choice adaptive tracking procedure. One of the three intervals, the target interval, contained the stimulus that was to be detected (i.e., the signal in the overshoot experiment and the probe in the suppression and adaptation-of-suppression experiments) with equal *a priori* probability. The task was to select the target interval by pressing the appropriate response button. Visual feedback was given after each trial indicating whether the participant had selected the correct or incorrect interval. The intervals were 272.5 ms long, cued visually, and separated by 500-ms gaps. The adaptive parameter was the signal level in the overshoot experiment and the probe level in the suppression and adaptation-of-suppression experiments. It was varied adaptively according to a two-down, one-up rule, which tracks 70.7% correct performance (Levitt, 1971). The steps were 10 dB up to the first reversal, 5 dB up to the second

reversal, and 2.5 dB for the remaining 8 reversals that made up each track. The last 6 reversals in each track were averaged to estimate threshold. For each condition and participant, tracks were run until the average of the last three threshold estimates had a standard error of less than 1.5 dB. The average of the last three threshold estimates was taken as the overall estimate.

In the measurements for setting the masker level, the stimulus to be detected was the signal and the adaptive parameter was the masker level. The masker level was varied according to a two-up, one-down tracking rule. In all other respects, the procedure was the same as stated above.

2.2.5 PARTICIPANTS

A total of seven participants (4 males and 3 females, aged between 20-28 years) were tested. They were screened for normal hearing (absolute thresholds ≤ 20 dB HL), had no reported history of audiological or neurological disease, and were not taking any neuroactive medication. Five participants took part in the original overshoot experiment, with the 2.5-ms signal duration, as well as in the suppression and adaptation-of-suppression experiments. Four participants (2 new) took part in the supplementary overshoot experiment, with the 12.5-ms signal duration. One participant took part in the piloting for this study, the others had no previous psychoacoustic experience.

Written informed consent was obtained from all participants. The experimental procedures were approved by the Ethics Committee of the Nottingham University School of Psychology and conformed to the guidelines of the Declaration of Helsinki at the time the data were collected (version 6, 2008), but were not formally pre-registered online in accordance with the 2014 amendment to the Declaration. Participants were paid at an hourly rate.

2.3 RESULTS

2.3.1 OVERSHOOT EXPERIMENT

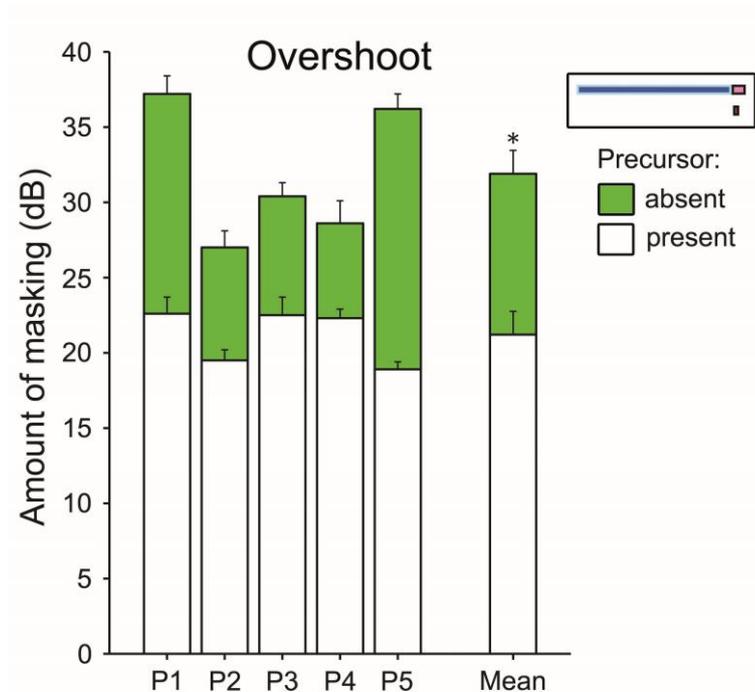


Figure 2.2: Individual (left bars) and average (right bars) signal detection thresholds from the overshoot experiment. The green bars in the background show the thresholds when the precursor was absent and the white bars in the foreground show the thresholds when the precursor was present. The overshoot is indicated by the visible portion of the green bars. All thresholds are expressed as amount of masking, that is, the masked threshold relative to the threshold in quiet. The error bars show the standard errors (SE). For the average, the SE was corrected for across-participant variability using the method proposed by Morey (2008). The stimulus configuration is shown in the inset (top right-hand corner).

In the overshoot experiment, all five participants showed substantially lower masked signal detection thresholds with than without the precursor present, indicating overshoot (Figure 2.2). The average overshoot amounted to 10.7 ± 2.2 dB (mean \pm standard error) and was statistically significant [paired *t*-test (2-tailed): $t(4) = 4.9, p = .008$]. The variation in the amount of overshoot across participants was considerable but consistent with previous studies (e.g., Strickland, 2004). The masker level was set individually for each participant so that it would just render the 25-dB SL signal used in the suppression and adaptation-of-suppression experiments inaudible (see Table 2.1). On average, the masker level was 76.6 ± 1.2 dB SPL. The precursor level was the same as the masker level (see Methods).

Participant	2.5-ms probe quiet threshold (dB SPL)	12.5-ms signal SPL level at 25 dB SL	Masker level (dB SPL)
P1	26	45	77
P2	33	53	75
P3	27	47	80
P4	32	53	78
P5	21	41	73
Mean ± SE	27.8 ± 2.2	47.8 ± 2.3	76.6 ± 1.2

Table 2.1: *Quiet detection threshold of the 2.5-ms probe (left column) and sound pressure level (SPL) of the 12.5-ms signal at 25 dB SL (middle column). The probe and 25-dB SL signal were used in the suppression and adaptation-of-suppression experiments. The right column shows the masker level needed to just render the 25-dB SL signal inaudible (used in all experiments). Individual and average values with standard errors (SEs) are shown in different rows.*

2.3.2 SUPPRESSION EXPERIMENT

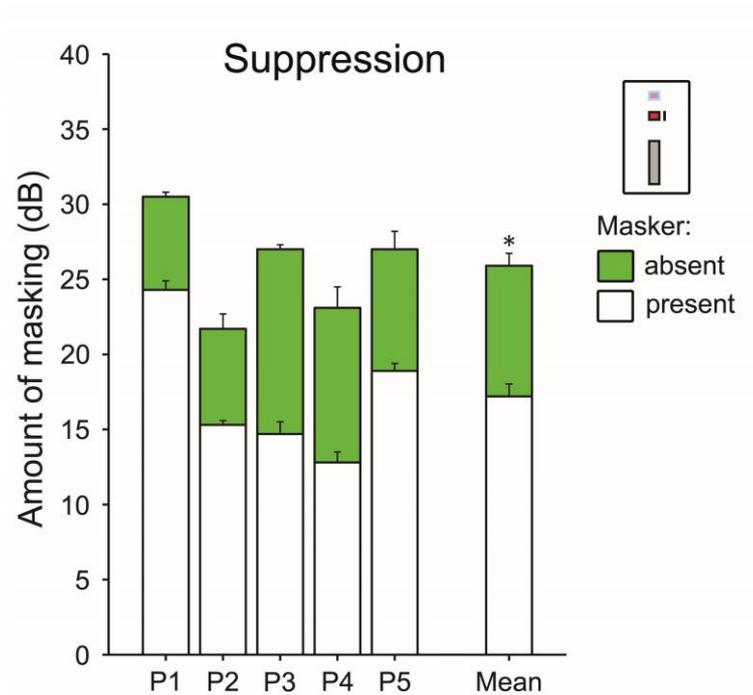


Figure 2.3: Individual (left bars) and average (right bars) probe detection thresholds from the suppression experiment. The green bars in the background show the thresholds when the probe was masked by the signal alone and the white bars in the foreground show the thresholds when it was masked by the signal and masker combined (see inset in right-hand corner). The visible portion of the green bars shows the suppression of the signal by the masker. As in Figure 2.2, all thresholds are expressed as amount of masking and the error bars show the SE (corrected for across-participant variability for the average).

Given that substantial overshoot was found in the overshoot experiment, and that, according to the adaptation-of-suppression model, overshoot is due to suppression being stronger when the precursor is absent than when it is present, it would be expected that, in the absence of the precursor, there would be substantial suppression. The suppression experiment showed that this was indeed the case. In all five participants, the signal caused considerably more forward masking when it was presented alone than when it was presented together with the masker (Figure 2.3). This suggests that the signal was being suppressed by the masker. On average, the probe detection threshold changed by 8.7 ± 1.2 dB; this was statistically significant [$t(4) = 7.4, p = 0.002$].

The amount of residual masking caused by the signal, when it was presented together with the masker and was thus inaudible, reveals whether the masker masked the signal exclusively by suppressive masking, or also by excitatory masking (Moore and Vickers, 1997; Plack et al., 2006). If the masker effect were exclusively suppressive, the signal response would be at the quiet threshold, and so, there should be little or no residual masking by the signal. If, on the other hand, the masker effect were exclusively excitatory, the signal should cause as much residual masking as when presented alone. The current data showed no significant residual masking [defined as the difference between the probe detection thresholds for the signal and masker combined and the masker alone, which was 2.1 ± 1.3 dB on average; $t(4) = 1.7, p = 0.172$]. This suggests that the masker effect on the signal was predominantly

suppressing. Despite causing mainly suppressive masking of the simultaneous signal, the masker alone caused considerable forward masking of the subsequent probe [15.5 ± 3.2 dB on average].

The suppression experiment involved a cue stimulus to disambiguate the signal from the probe. A control measurement showed that the cue itself did not cause any significant masking of the probe [average amount of masking by the cue = 0.9 ± 0.7 dB, which was not statistically significant; $t(4) = -0.9, p = 0.204$].

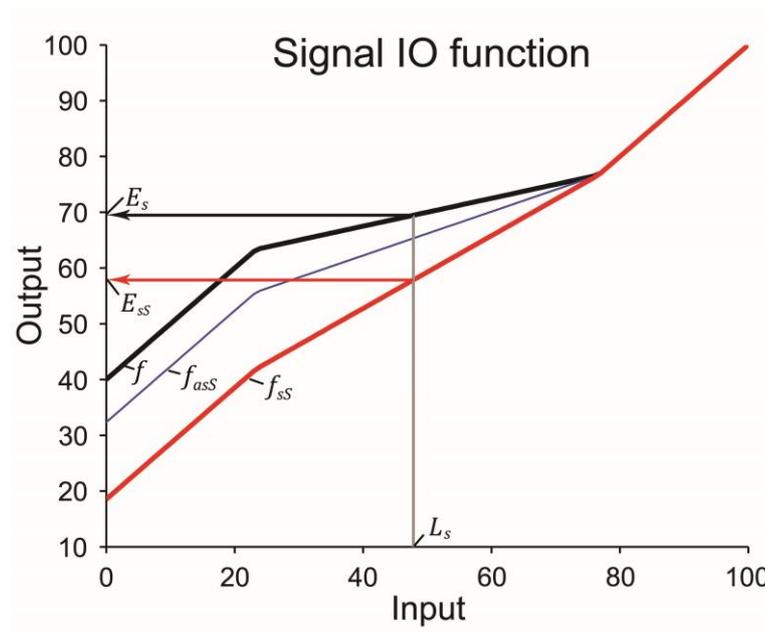


Figure 2.4: Simulated cochlear IO functions of the signal in the suppression experiment. The bold black line, labelled f , shows the IO function when the signal is presented alone and is thus unsuppressed. The bold red line, labelled f_{sS} , shows the IO function when the signal is presented together with the masker and is thus suppressed. The grey

vertical line shows the average sound pressure level of the signal. The horizontal black and red arrows show the response levels of the unsuppressed (E_S) and suppressed signal (E_{SS}), respectively. The blue line, labelled f_{asS} , shows the simulated signal IO function in the presence of both the masker and precursor (to be discussed in the adaptation-of-suppression experiment). In this example, it was assumed that all of the measured overshoot was caused by adaptation of suppression. The model results shown here are based on the averaged data across participants and are for illustration only. The model predictions presented in the text are based on the individual data.

The difference between the probe detection thresholds for the signal alone and for the signal and masker combined might be much larger than the actual suppression in cochlear gain exerted by the masker. This is because the cochlear IO function of the probe is compressive for mid-range levels and so, a small change in gain might yield a larger change in probe detection threshold. Here, an established model of the cochlear IO function was used to convert the change in probe detection threshold into an estimate of the actual change in cochlear gain. The model assumes that the cochlea applies active amplification at, and within a narrow range around, the characteristic frequency, but not at more remote frequencies (Rhode, 1971). It is assumed that the amplification is maximal at low sound levels, decreases progressively at medium sound levels and is absent at high sound levels. The amplification is assumed to

apply instantaneously. This general model has been successfully used to fit psychophysical estimates of the auditory filter widths as a function of sound level (Glasberg and Moore, 2000) and to derive psychophysical estimates of cochlear compression using various types of experimental paradigms (e.g., Plack and Arifianto, 2010; Yasin et al., 2013b).

In the current implementation of this model, which is similar to the one used by Yassin and Plack (2003), the cochlear IO function, f , was expressed as the sum of the sound level, L , and a level-dependent gain, $G(L)$; in units of intensity: $f(L) = 10^{(L+G(L))/10}$ (Figure 2.4, black line). At low sound levels up to a first break point, BP_1 , the gain was assumed to be constant at the maximum value G_{max} :

$$G(L \leq BP_1) = G_{max} \quad (1).$$

Between BP_1 , and a second break point, BP_2 , the gain was assumed to decrease linearly from G_{max} to zero, at a rate of $1 - c$, where c is the compression exponent:

$$G(BP_1 \leq L \leq BP_2) = (c - 1)(L - BP_1) + G_{max} \quad (2).$$

The compressive range was assumed to be symmetric about $L = 50$ dB SPL, so $BP_1 = 50 - G_{max}/2(1 - c)$ and $BP_2 = 50 + G_{max}/2(1 - c)$.

Above BP_2 , the gain was assumed to be zero:

$$G(L \geq BP_2) = 0 \quad (3).$$

Based on psychophysical data from humans (Nelson et al., 2001; Yasin et al., 2013b) and physiological data from chinchillas (Ruggero et al., 1997), G_{max} was set to 40 dB and c was set to 0.25. This meant that BP_1 and BP_2 were equal to 23.3 and 76.7 dB SPL, respectively. f was used to calculate the masking effect, W , of the signal alone (W_S), the masker alone (W_M) and the signal and masker combined (W_{SM}); the masking effect corresponds to the cochlear response to the probe at the respective probe detection threshold, L_{Probe} : $W = f(L_{Probe})$. The residual masking effect of the suppressed signal was calculated as the difference between the masking effects of the masker alone and the masker and signal combined: $W_{SS} = W_{SM} - W_M$. W_{SS} was used to calculate the cochlear response to the suppressed signal, E_{SS} , by assuming that E_{SS} and W_{SS} are related through a constant factor, k , which represents the signal-to-noise ratio at masked threshold: $E_{SS} = W_{SS}/k$. k was derived from the masking effect of the signal alone, W_S , and the cochlear response to the signal alone, E_S : $k = W_S/E_S$. E_S was calculated by passing the signal level, L_S (47.8 ± 2.3 dB SPL; see Table 2.1), through the cochlear IO function, f . k was equal to 1.48 ± 0.15 , on average. The suppressed signal excitation, E_{SS} , was used to calculate the cochlear gain of the suppressed signal, G_{SS} , by subtracting the signal level, L_S : $G_{SS} = 10 \log_{10}(E_{SS}) - L_S$. The suppression is equal to the gain of the signal alone, $G(L_S)$, minus the gain of the suppressed signal, G_{SS} . The observed suppression depends, not just on the effectiveness of the suppressor (i.e., the masker in this case), but also on the level of the suppressor (i.e., the signal); the higher the

suppressee's level, the lesser its cochlear gain, and so, the lesser the suppression. A better measure of the suppressor's effectiveness is thus its effect on the maximum cochlear gain, G_{max} . In order to calculate the suppression in G_{max} , the IO function of the suppressed signal, f_{SS} (Figure 2.4, red line) was calculated. For that, the squared difference between $f_{SS}(L_s)$ and the response to the suppressed signal, E_{SS} , was minimised by varying G_{max} using *lsqnonlin* in Matlab. The breakpoints were kept constant, and so, varying G_{max} also varied the compression exponent, c . The model was applied to the data from each participant separately.

According to this model, the masker suppressed the cochlear gain of the signal by, on average, 12.7 ± 2.8 dB. Figure 2.4 shows the cochlear IO function of the suppressed signal, f_{SS} (red line). The maximum gain of this IO function was equal to 16.6 ± 4.3 dB, on average, suggesting that the masker suppressed the maximum cochlear gain by 23.3 dB, or 58%. As a result, the compression exponent increased from 0.25 to 0.69 ± 0.08 .

In order to test how sensitive the estimated suppression was to the parameters of the cochlear IO function, f , the model was re-ran with a range of values for the maximum cochlear gain, G_{max} , and compression exponent, c . G_{max} and c were varied orthogonally, with G_{max} ranging from 30 to 50 dB in 5-dB steps, and c ranging from 0.15 to 0.35 in steps of 0.05. Over these ranges, the estimated average suppression in the signal's cochlear gain ranged between 11.32 to 14.73 dB. It has been suggested that a change in the maximum cochlear gain, G_{max} , does not

change the maximum compression, c , but instead, changes the lower bound of the compressive range, BP_1 (Plack et al., 2004). Using this assumption, instead of the assumption that the breakpoints remain fixed, did not affect the estimated suppression.

2.3.3 ADAPTATION-OF-SUPPRESSION EXPERIMENT

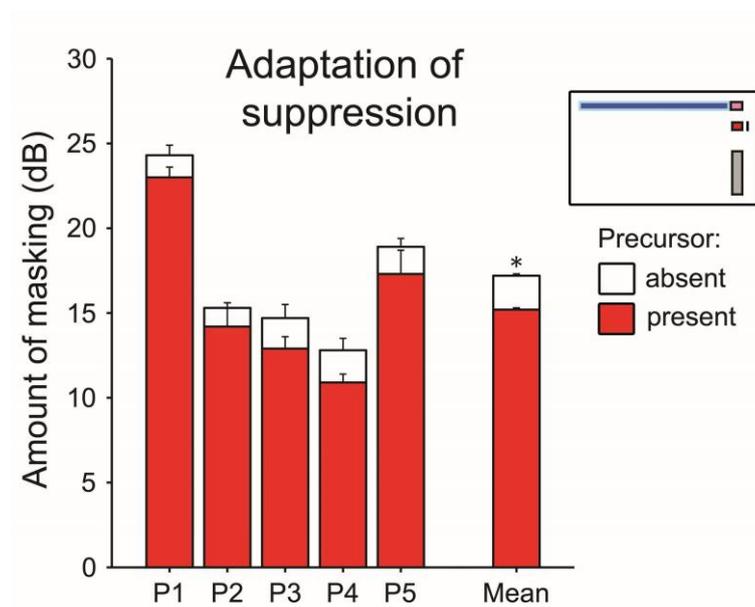


Figure 2.5: Individual (left bars) and average (right bars) probe detection thresholds from the adaptation-of-suppression experiment. The white bars in the background show the thresholds when the precursor was absent and the red bars in the foreground show the thresholds when it was present (see inset in right-hand corner). As in Figure 2.2, all thresholds are expressed as amount of masking, and the error bars show the SE (corrected for across-participant variability for the average).

According to the adaptation-of-suppression model of overshoot, the precursor should have reduced the suppressive masking of the signal by the masker. This should have increased the response to the signal and thus its forward-masking effect on the probe, causing an increase in probe detection threshold. This, however, was not observed. Instead of an increase, the precursor caused a small (1.5 ± 0.2 dB on average) but significant [$t(4) = -10.2, p = 0.001$] decrease in probe detection threshold (Figure 2.5). A similar (0.9 ± 0.9 dB on average) albeit non-significant [$t(4) = -1.0, p = 0.359$] decrease in the probe detection threshold due to the precursor was also observed in the control experiment, where the probe detection threshold was measured in the presence of the masker alone, or the masker and precursor combined. The precursor effects in the adaptation-of-suppression and control experiments were not significantly different from one another [$t(4) = -0.6, p = 0.573$].

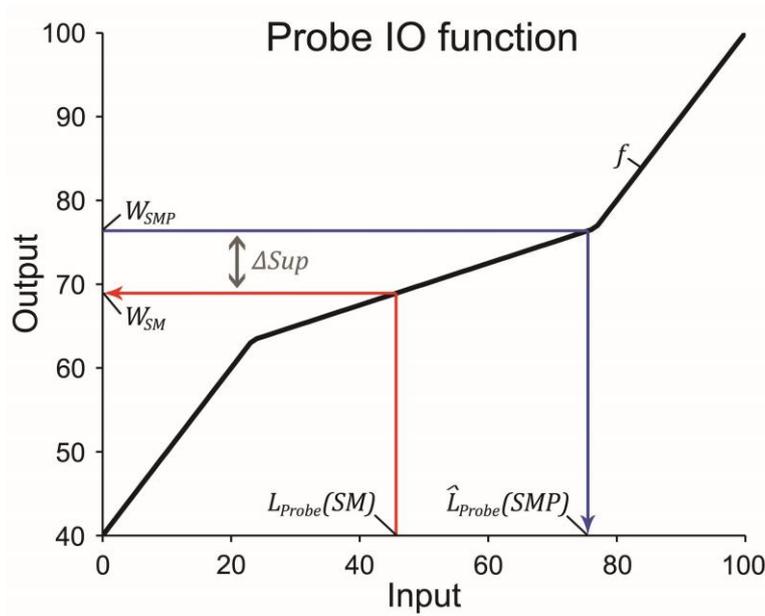


Figure 2.6: Simulated cochlear IO function, f , of the probe (bold black line) in the suppression and adaptation-of-suppression experiments. The red arrow connects the measured detection threshold, $L_{\text{Probe}}(\text{SM})$ (input), and the simulated response level, W_{SM} (output), of the probe when it is masked by the signal and masker combined. The blue arrow connects the simulated response level, W_{SMP} , and predicted detection threshold, $\hat{L}_{\text{Probe}}(\text{SMP})$, of the probe when it is masked by the signal, masker and precursor. In this example, it was assumed that all of the measured overshoot was caused by adaptation of suppression.

Does the failure to obtain an increase in probe detection threshold in the adaptation-of-suppression experiment represent a false negative outcome? A false negative outcome might have arisen if any precursor-induced increase in the cochlear gain of the signal (adaptation of suppression) produced a greater overshoot effect than it did a change in

the probe detection threshold in the adaptation of suppression experiment. In order to test this possibility, the same model of the cochlear IO function as in the suppression experiment was used to estimate the amount of change in probe detection threshold that would be expected for a given amount of overshoot caused by adaptation of suppression, O_{as} . Any overshoot caused by adaptation of suppression would be associated with an increase in the cochlear gain of the signal. The remaining overshoot, O_c , would be assumed to be due to release from central masking effects, such as transient masking or attentional diversion, and thus not be associated with any change in cochlear gain. The control experiment showed that the masker and precursor combined caused the same amount of forward masking as the masker alone, indicating that the precursor caused no more excitatory masking than the masker alone. This suggests that the measured overshoot, O_{meas} , reflects all of the overshoot that actually occurred, rather than some of the overshoot being counteracted by additional excitatory masking by the precursor. Thus,

$$O_{meas} = L_S(M) - L_S(MP) = O_{as} + O_c \quad (4);$$

here, $L_S(M)$ and $L_S(MP)$ are the signal detection thresholds for the masker alone and the masker and precursor combined. O_{as} was varied from zero to O_{meas} , in 1-dB steps. First, the increase in the maximum cochlear gain of the signal, G_{max} , associated with a given adaptation of suppression-related overshoot, O_{as} , was calculated by calculating the

cochlear IO function of the signal after adaptation of suppression, f_{asS} . For that, the squared difference between the signal response with both central and adaptation of suppression-related overshoot taken into account, $f_{asS}(L_S(M) - O_{as} - O_c) = f_{asS}(L_S(MP))$ [see Eq. (4)], and the signal response with only central overshoot taken into account, $f_{sS}(L_S(M) - O_c)$, was minimised. Here, f_{sS} is the cochlear IO function of the signal when it is fully suppressed by the masker, which was derived in the suppression experiment. In equating $f_{asS}(L_S(MP))$ and $f_{sS}(L_S(M) - O_c)$, it was assumed that any increase in cochlear gain caused by adaptation of suppression would counteract the corresponding decrease in signal detection threshold, O_{as} , to create a constant signal response at threshold. As in the suppression experiment, G_{max} was varied using *lsqnonlin* in Matlab, whilst keeping the breakpoints, BP_1 and BP_2 , fixed [see Eqs (1)-(3)]. The dashed blue line in Figure 2.4 shows f_{asS} if *all* of the measured overshoot had been caused by adaptation of suppression. The actual gain of the signal in the adaptation-of-suppression experiment was then calculated when both the masker and precursor were present, $G_{asS} \cdot G_{asS}$ by substituting the signal level used in the adaptation-of-suppression experiment, L_S , for L , and the maximum cochlear gain after adaptation of suppression, $G_{max}(asS)$, for G_{max} in Eqs (1)-(3). G_{asS} was used to estimate the masking effect of the signal with both the masker and precursor present, W_{SMP} . W_{SMP} was assumed to be equal to the masking effect of the signal with only the masker present, W_{SM} , times a factor representing the

increase in the signal gain as a result of adaptation of suppression, $\Delta Sup = G_{asS} - G_{SS}$: $W_{SMP} = W_{SM} \cdot 10^{\Delta Sup/10}$ (Figure 2.6). The expected probe detection threshold with the signal, masker and precursor present, $\hat{L}_{Probe}(SMP)$, was then estimated by passing W_{SMP} through the inverse of the cochlear IO function, f , (Figure 2.6, blue arrow). As in the suppression experiment, the model was applied to each participant's data separately, and the results were averaged.

According to this model, every 1-dB increase in overshoot caused by adaptation of suppression would have been expected to be associated with an increase in probe detection threshold of, on average, 2.57 ± 0.15 dB in the adaptation-of-suppression experiment.

As in the suppression experiment, the model was re-ran with a range of values for the maximum cochlear gain, G_{max} , and compression exponent, c . Again, G_{max} was varied between 30 and 50 dB in 5-dB steps, and c was varied between 0.15 and 0.35 in steps of 0.05. Over these ranges, the rate of increase in probe detection threshold with every 1-dB increase in adaptation of suppression-related overshoot ranged between 1.72 and 3.50 dB, on average. Using the assumption that the lower breakpoint, BP_1 , changes when G_{max} changes, rather than the compression exponent, c , increased the rate of increase in probe detection threshold per 1-dB increase in adaptation of suppression-related overshoot from 2.57 dB to 3.31 (± 0.42) dB.

2.3.4 SUPPLEMENTARY OVERSHOOT EXPERIMENT

The signal was longer in the adaptation-of-suppression experiment (12.5 ms) than in the original overshoot experiment (2.5 ms). It is possible that the longer signal was less affected by the precursor than the shorter one, which would explain why overshoot was found but no adaptation of suppression. In order to test this possibility, a “supplementary overshoot experiment”, which measured overshoot for both the 2.5- and 12.5-ms signals, was conducted. The procedures were the same as in the original overshoot experiment. Four participants (2 from the original group and 2 new) took part in this experiment.

On average, 7.2 ± 1.9 dB of overshoot was found for the 2.5-ms signal, compared to 5.7 ± 0.8 dB for the 12.5-ms signal. This difference was not significant [paired t-test: $t(3) = 0.9$, $p = 0.410$]. The overshoot for the 2.5-ms signal was smaller than that measured in the original overshoot experiment (10.7 ± 2.2 dB), albeit non-significantly [unpaired t-test: $t(7) = 2.0$, $p = 0.091$]. Given that the stimuli were identical, this difference would appear to be due to variability between participants. The fact that the 2.5- and 12.5-ms signals yielded similar overshoot rules out the possibility that the failure to obtain adaptation of suppression was due to the difference in signal duration between the original overshoot and adaptation-of-suppression experiments.

2.4 DISCUSSION

The adaptation-of-suppression model of overshoot posits that off-frequency overshoot arises because the precursor reduces suppressive masking of the signal by the masker. The reduction in suppression is thought to arise as a result of a reduction in the cochlear amplification of the masker (adaptation of suppression), which is thought to be mediated by the MOCR. In order to test this model, overshoot, suppression, and adaptation of suppression were measured using similar stimuli in the same set of participants. Substantial overshoot and suppression was found in all participants, with effect sizes similar to those found in previous studies (e.g., Bacon and Moore, 1986; Lee and Bacon, 1998). Despite this, no evidence of adaptation of suppression, that is, no precursor-induced increase in probe detection threshold in the adaptation-of-suppression experiment, was found. This suggests that adaptation of suppression did not appreciably contribute to the observed overshoot effect. Predictions based on an established model of the cochlear IO function showed that, due to the nonlinearity of the cochlear IO functions of the signal and probe, every decibel of overshoot caused by adaptation of suppression would have been associated with a precursor-induced increase in probe detection threshold of around 2.5 dB in the adaptation-of-suppression experiment. Thus, even if only 1 dB of the observed overshoot had been caused by adaptation of suppression, the precursor should have caused a detectable increase in probe detection threshold. Instead, it caused a small but significant decrease in

probe detection threshold. A similar precursor effect was found in the control experiment, where the signal was not present. This suggests that the precursor effect in the adaptation-of-suppression experiment was caused by a central mechanism. The results of Scharf et al. (2008) suggest that the masker onset would have diverted attention away from the probe frequency towards the masker frequency and that the precursor would have mitigated this effect by giving the listener time to refocus attention back to the probe frequency.

There are three possible reasons as to why the precursor did not cause any measurable adaptation of suppression. Firstly, the precursor may not have caused any reduction in the cochlear amplification of the masker, either because it was spectrally too narrow to elicit the MOCR, or because the MOCR effect did not occur at the precursor (and thus masker) frequency. In studies using OAE measurements, a tone presented contralateral to the probe has been found to be a poor elicitor of the MOCR (e.g., Berlin et al., 1993; Lisowska et al., 2002; Lilaonitkul and Guinan, 2009b), but an ipsilateral tonal elicitor (like the precursor used in the current study) has been found to be more effective (Lilaonitkul and Guinan, 2009b). However, whether presented contralateral or ipsilateral to the probe, noise elicitors have been shown to be far more effective than tones (Lilaonitkul and Guinan, 2009a, b). It is thus plausible that the MOCR was not sufficiently elicited by the tonal precursor to cause a measurable reduction in suppressive masking.

A second possible reason that the precursor did not reduce suppressive masking is that, although the MOCR was elicited, it did not have a significant effect at the masker frequency. Lilaonitkul and Guinan (2012) found that, for a 1-kHz probe, the most effective elicitor was around 0.5–1 octave below the probe frequency, but that for a 4-kHz probe (closer to the frequency of the masker and precursor used in the current study) the most effective elicitor was at, or slightly above, the probe frequency. However, it is possible that the masker was not susceptible to a cochlear-gain reduction because it was presented at a high level in both the overshoot and suppression experiments (77 dB SPL, on average), and was therefore not subject to a significant amount of cochlear gain (see Chapter 1, Section 1.1.1).

Finally, the precursor may have elicited the MOCR and caused a reduction in the masker gain, but this may not have reduced the suppression exerted by the masker. This latter scenario is predicted by models which assume that there is no interaction between different frequency channels that have independent active processes at the level of cochlea, like the dual-filter model of suppression, which is described by Plack et al. (2002). The dual-filter model of cochlear frequency-selectivity describes the response of each point along the cochlear partition as the combination of two (tip and tail) filters (Goldstein, 1989; Meddis et al., 2001). The tip filter simulates the amplified peak, and the tail filter the passive tail, of the cochlear response. The tail filter is broader than the tip filter and centred at a slightly lower frequency. In

the model used by Plack et al., suppression occurs within the tip filter. This means that the amount of suppression is determined by the sound level of the suppressor and thus not influenced by its cochlear gain. Plack et al. (2002)'s version of the dual-filter model assumes the existence of a third filter, which, unlike the tip and tail filters, is not intended to simulate the cochlea response. This third filter is similarly broad as the tail filter, but centred at a frequency slightly *above* the tip filter. It enables the model to reproduce the difference in suppression threshold between low- and high-side suppressors (e.g., Shannon, 1976; Cooper, 1996). However, the model fails to predict the difference in the growth rate of suppression with suppressor level between low- and high-side suppressors (e.g., Duifhuis, 1980; Delgutte, 1990). Another approach to implement suppression in a dual-filter model, proposed by Goldstein (1990), does predict the difference in the suppression growth rate between low- and high-side suppressors. In Goldstein's model, the suppressor and suppressee are processed through the same filter and are thus subject to the same cochlear gain. This means that any reduction in cochlear gain by the MOCR would affect the suppressor and suppressee equally, and so, as in Plack et al.'s model, little or no net effect on the amount of suppression would be expected. Like Plack et al.'s model, Goldstein's model has to include a component with no intended physiological correlate; in this case, an expansive nonlinearity in the tail filter. This is to counter the effect of a compressive nonlinearity, which is applied to the combined tip and tail filter responses in order to produce

suppression. While Goldstein's model reproduces the difference in the growth rate of suppression between low- and high-side suppressors, it does not predict the finding, from both physiological and psychophysical studies, that maximum suppression occurs at a frequency above, rather than at, the frequency of the suppressee (Arthur et al., 1971; Shannon, 1976; Duifhuis, 1980; Cooper, 1996). This finding has led to the hypothesis that the active process (i.e., the group of OHCs) that amplifies the cochlear response is located basal to the response peak (see Patuzzi, 1996). The suppressor is assumed to "jam" the suppressee's active process. The effectiveness of this jamming is assumed to be determined by the size of the suppressor response at the place where the suppressee's active process is located. This physiological model of suppression also explains the differences in both suppression threshold and suppression growth rate between low-and high-side suppressors. Transmission-line models, which try to emulate the physiological properties of the cochlea, correctly capture all the salient properties of suppression without any further assumptions (Epp et al., 2010). In transmission-line models, suppression arises as a result of interactions between different frequency channels with independent active processes. As a result, transmission-line models would be expected to predict that reducing the gain of a high-side suppressor, for instance through elicitation of the MOCR, would reduce the amount of suppression caused.

Irrespective of why the precursor did not cause the probe detection threshold in the adaptation-of-suppression experiment to increase, the fact that it did not suggests that the observed overshoot was not associated with any appreciable increase in the signal response. In contrast, for enhancement, there is clear evidence that the precursor causes the signal response to increase (Viemeister and Bacon, 1982; Byrne et al., 2011). However, this does not necessarily imply that enhancement is caused by adaptation of suppression. In fact, Wright et al. (1993) and Wright (1996) produced evidence against the adaptation-of-suppression account of enhancement. They measured suppression and enhancement in same set of participants and found a negative correlation between them; the adaptation-of-suppression hypothesis would predict a positive correlation. Furthermore, Viemeister and Bacon found, albeit with few participants, that the signal response was enhanced by a similar amount irrespective of whether the masker was actually present. Although Viemeister and Bacon speculated otherwise, it would generally be assumed that, when the masker was absent, the signal would not have been suppressed, and so, the observed enhancement could not have been caused by adaptation of suppression.

It is possible that enhancement is caused, not by adaptation of suppression, but, rather, by an increase in the responsiveness of the frequency channels within the spectral complement of the precursor. Thus, the mechanism of enhancement may be related to the mechanism underlying the Zwicker tone (Zwicker, 1964; Lummis and Guttman,

1972). The Zwicker tone is a faint tonal sensation following the presentation of a spectrally notched precursor similar to those used to produce enhancement, with a pitch in the range of the precursor notch. Wiegrebe et al. (1996) reported evidence suggesting that the Zwicker tone arises as a result of an increase in auditory responsiveness at frequencies corresponding to the Zwicker-tone pitch; at these frequencies, absolute hearing sensitivity was increased following the precursor presentation. The amount of increase in hearing sensitivity (up to 13 dB) was similar to the amount of increase in the signal response as a result of enhancement (Viemeister and Bacon, 1982; Byrne et al., 2011). The Zwicker tone is an inconspicuous percept and can only be elicited at low or medium precursor levels. As a result, the precursor levels used by Wiegrebe et al. were much lower than those used in many enhancement experiments. Thibodeau (1991) showed that robust enhancement occurs up to very high precursor levels (91 dB SPL). It is currently not known whether the increase in absolute hearing sensitivity found by Wiegrebe et al. is limited to low and medium precursor levels like the Zwicker tone, or whether it persists at high precursor levels like enhancement. The properties of the Zwicker tone suggest that it arises centrally rather than peripherally. For instance, it is impossible to produce beating between the Zwicker tone and an external tone of a similar frequency (Krump, 1993; cited in Wiegrebe et al., 1996) and the Zwicker tone does not interact with spontaneous otoacoustic emissions (Wiegrebe et al., 1996). There is evidence suggesting that enhancement

also arises centrally. Physiological studies have found enhancement in single-neuron responses in the inferior colliculus (Nelson and Young, 2010) but not in the auditory nerve (Palmer et al., 1995). Psychophysical findings suggest that enhancement occurs, at least in part, beyond the point where the monaural pathways converge (Serman et al., 2008; Carcagno et al., 2012). More extensive characterisation is needed to better understand the relationship between the two phenomena.

Enhancement causes an increase in the signal response, which, like the Zwicker tone, might be caused by an increase in auditory responsiveness within the spectral complement of the precursor, and which manifests as an increase in the signal's forward-masking effectiveness. In contrast, in overshoot, any precursor-induced increase in auditory responsiveness would not be expected to change the signal's forward-masking effectiveness. This is because of the short signal durations used in overshoot experiments. Any increase in auditory responsiveness would likely outlast the signal and equally affect the probe, leaving the probe detection threshold unchanged. The current study included a control experiment, which measured the probe detection threshold in the presence of the masker alone and the masker and precursor combined. Any precursor-induced increase in responsiveness to the probe should have resulted in a decrease in probe detection threshold. This however, was not observed.

The results of this study indicate that overshoot associated with a precursor and masker with energy only above the signal frequency is likely based on a different mechanism than enhancement. It is possible that enhancement only occurs when the precursor contains energy both below and above the signal frequency. This has been shown to be the case for the Zwicker tone, which is abolished when the lower and upper bands of the precursor are presented to different ears (Krump, 1993; cited in Wiegrebe et al., 1996). Precursor and masker energy above the signal frequency accounts for the majority of off-frequency overshoot (Schmidt and Zwicker, 1991). It is possible that off-frequency overshoot is caused by transient masking. Transient masking refers to the perceptual confusion between two transient events that occur close together in time (the short signal and masker onset in the case of overshoot; Bacon and Moore, 1987). Alternatively, off-frequency overshoot may be caused by attentional diversion. Scharf et al. (2008) found evidence suggesting that the masker onset diverts attention away from the signal frequency towards the masker frequency, and that the precursor mitigates this effect by allowing the listener to refocus attention back to the signal frequency. Both transient masking and attentional refocusing are higher-level effects, which, unlike enhancement, are unlikely to have correlates in subcortical processing.

CHAPTER 3

A QUANTITATIVE MEASURE OF THE CONTRALATERAL MEDIAL OLIVOCOCHLEAR REFLEX

3.1 INTRODUCTION

In Chapter 2, it was found that overshoot is unlikely to be a perceptual consequence of the MOCR, and overshoot is therefore not pursued further in this chapter. Instead, the aim of this chapter is to measure MOCR-induced cochlear-gain reduction quantitatively by modifying a psychophysical method for measuring cochlear gain and compression directly.

The current study followed a similar approach to four previous studies, where cochlear gain was measured psychophysically using the “temporal masking curve” (TMC) method (Krull and Strickland, 2008; Roverud and Strickland, 2010; Aguilar et al., 2013) or variants thereof (Yasin et al., 2014). The TMC method (Nelson et al., 2001) involves measuring the level of a forward masker needed to just mask a short sinusoidal signal as a function of the temporal gap between the masker and signal. Crucially, the masker frequency is either at or well below the signal frequency (“on/off frequency”). The masking effect of the on-frequency masker is caused by the peak of the cochlear response, which is amplified by the cochlear gain, whereas the masking effect of the off-frequency masker is caused by the basal tail of the response, which is unaffected by gain (Robles and Ruggero, 2001). Thus, the difference between the on- and off-frequency masking effects should reflect the amount of amplification of the peak response. The previous studies measured TMCs both with and without an MOCR elicitor. In different studies, the elicitor was presented either contralateral (Aguilar et al., 2013) or ipsilateral (Krull and Strickland, 2008; Roverud and Strickland, 2010; Yasin et al., 2014) to the signal and masker. An ipsilateral elicitor activates the crossed medial olivocochlear fibres, whereas a contralateral elicitor activates the uncrossed fibres (Guinan, 2006). In either case, the elicitor would be expected to reduce the cochlear gain of the masker response, the signal response, or both, and thereby alter the masker level at threshold (i.e., the masker level needed to just mask the

signal). However, the elicitor might also cause some direct masking of the signal, through mechanisms unrelated to cochlear gain (e.g., neural swamping), particularly when the elicitor and signal are presented ipsilaterally. Yasin et al. (2014) controlled for this possibility by increasing the signal level so that the signal would be equally detectable with or without the elicitor. In the current study, the approach of the earlier three studies (Krull and Strickland, 2008; Roverud and Strickland, 2010; Aguilar et al., 2013), which used the same low signal level for both the with- and without-elicitor conditions, was followed. It was shown that, even with the signal level fixed, it is still possible to control for direct masking of the signal by the elicitor, because direct masking and cochlear-gain reduction produce distinct patterns of effects on the on-frequency TMC. The previous three studies only measured off-frequency TMCs. Here, it is shown that, in the off-frequency TMC, the effects of direct masking and cochlear-gain reduction are indistinguishable. The psychophysical TMC measurements are compared with physiological measurements of contralateral OAE suppression using the same elicitor and participants.

3.2 EXPERIMENT 1: TEMPORAL MASKING CURVE

(TMC) MEASUREMENTS

3.2.1 GENERAL OUTLINE AND EXPECTATIONS

In this experiment, TMCs were measured for a short (5-ms) sinusoidal signal at 2 kHz, presented at a fixed level of 10 dB SL above its quiet threshold. The masker was also sinusoidal, and its frequency was either the same as (Figure 3.1A), or well below (1.22 kHz; Figure 3.1B), the signal frequency. The masker duration was much shorter (25 ms) than in the original TMC measurements (Nelson et al., 2001) to minimise the extent to which the masker itself would be able to elicit the MOCR in time to affect the signal detectability (James et al., 2002a; Backus and Guinan, 2006). The TMCs were measured either with or without a broadband-noise elicitor, presented contralateral to the signal and masker. In order to avoid eliciting the MEMR, the elicitor was presented at a conservative level below the lowest MEMR threshold across participants. In the following, expectations are derived about how the on- and off-frequency TMCs would be affected if the elicitor causes cochlear-gain reduction or direct masking (Figure 3.1).

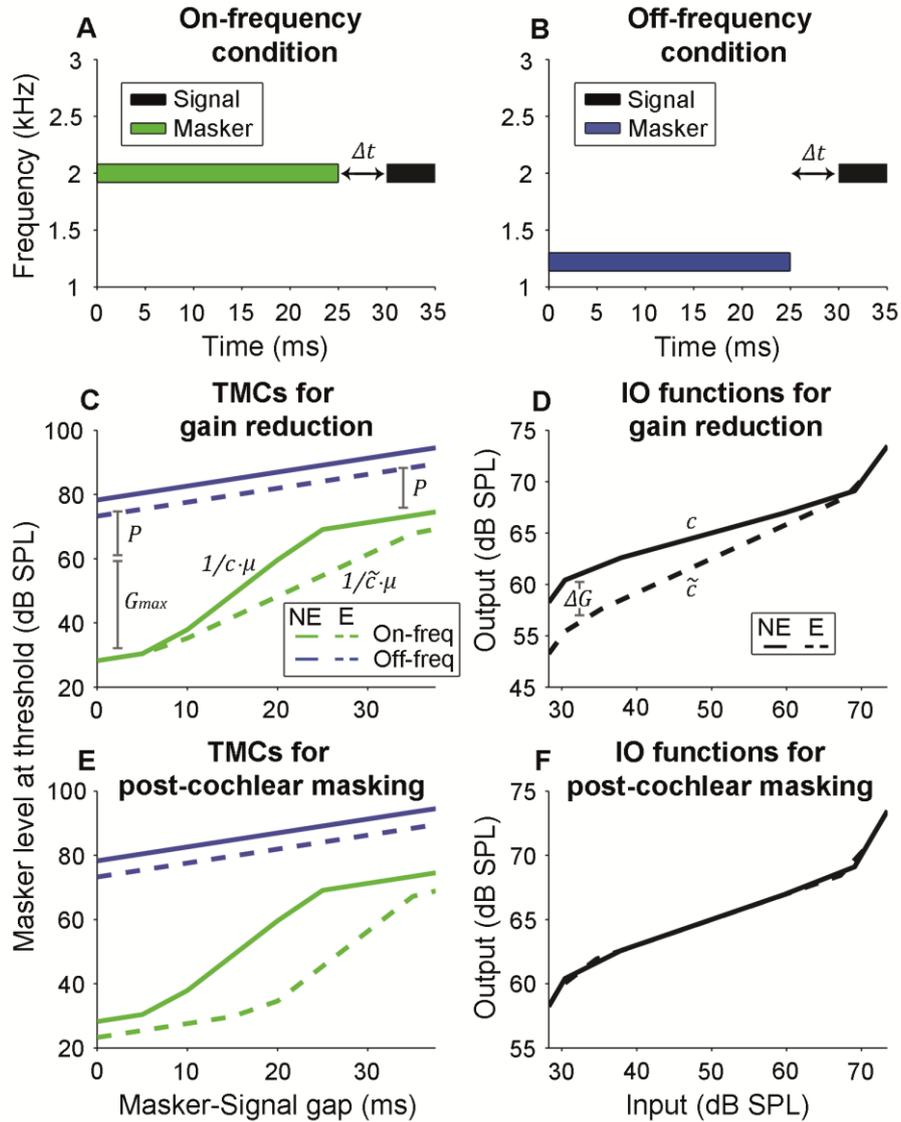


Figure 3.1: Panels A and B show a schematic representation of the spectral and temporal characteristics of the stimuli for the on- and off-frequency conditions. The signal and masker are represented by different colors (see legend) and the masker-signal gap is labelled Δt . Panels C and E show simulated TMCs for the on- and off-frequency conditions (green and blue, respectively) both with (dotted lines) and without (solid lines) the elicitor. Panels C and D show simulated TMCs with and without an elicitor (E and NE, respectively), assuming that the elicitor caused only a reduction

in cochlear gain (C) or only post-cochlear masking (D). In panel C, it is shown that the difference between the on- and off-frequency masker level at threshold is due both to passive attenuation of the basilar membrane response to the off-frequency masker (P) and to cochlear gain increasing the response to the on-frequency masker (G_{max}). The rate of growth for the on-frequency TMC without the elicitor at intermediate masker levels is equal to $1/c \cdot \mu$, where c is the compression exponent without the elicitor and μ is the decay rate of the masker response over time (i.e., the slope of the off-frequency TMC). The growth rate with the elicitor is equal to $1/\tilde{c} \cdot \mu$, where \tilde{c} is the compression exponent with the elicitor present. Panels D and F show the IO functions derived from the TMC estimates in panels C and E. For these IO functions, the rate of growth in the output at intermediate input levels is equal to the compression exponent, c . The difference in output between the with- and without-elicitor conditions is equal to the amount of gain reduction (ΔG).

The TMC method relies on the assumption that, at the place along the basilar membrane that responds maximally to the signal, the rate of decay is independent of the masker frequency (Nelson et al., 2001). Thus, under the assumption that the basal tail response to the off-frequency masker grows linearly with increasing masker level, the off-frequency masker level at threshold should increase linearly with increasing temporal gap between the masker and signal, and the rate of increase should correspond to the rate of decay of the masker response, μ (solid blue line in Figure 3.1C). Physiological studies have shown that cochlear

gain is maximal at low sound levels, decreases with increasing level for intermediate levels, and is zero at high levels (Robles and Ruggero, 2001). As a result, the IO function of the peak response to the on-frequency masker will grow linearly (with a slope of unity) at low and high masker levels, but compressively (with a slope, c , less than unity) at intermediate levels (solid black line in Figure 3.1D). This means that the on-frequency TMC (solid green line in Figure 3.1C) will have the same slope as the off-frequency TMC at short and long masker-signal gaps, but at intermediate gaps, the slope of the on-frequency TMC will be steeper by a factor corresponding to the reciprocal of the compressive IO-function slope, $1/c$. At short masker-signal gaps, the difference between the on- and off-frequency masker level at threshold would be assumed to reflect the sum of the active amplification of the peak response to the on-frequency masker (G_{max} ; Figure 3.1C) and the passive attenuation of the tail response to the off-frequency masker (P ; Figure 3.1C). Towards longer masker-signal gaps, the difference decreases to the passive attenuation only.

If the elicitor causes a reduction in cochlear gain, the off-frequency masker level at threshold should decrease equally at all masker-signal gaps, and the amount of decrease should be equal to the amount of gain reduction, ΔG (compare solid and dashed blue lines in Figure 3.1C). This is because the signal response would be diminished by ΔG , but the tail response to the off-frequency masker would be unchanged. For the on-frequency masking condition, the effect of a gain reduction by the elicitor

would be expected to depend on the masker-signal gap. At short gaps, the gain reduction would affect the signal and masker responses equally, and so, there should be no change in the masker level at threshold (compare solid and dashed green lines in Figure 3.1C). At intermediate gaps, the on-frequency TMCs with and without the elicitor should diverge. This is because a reduction in gain would make the IO function of the cochlear peak response grow less compressively (with a slope $\tilde{c} > c$; compare solid and dashed lines in Figure 3.1D), leading the slope of the on-frequency TMC to become shallower (by a factor c/\tilde{c} ; Figure 3.1C). At long masker-signal gaps, the on-frequency masker response turns passive, and so, the difference between the on-frequency masker level at threshold with and without the elicitor will, like the difference between the off-frequency masker levels at threshold, become equal to the amount of gain reduction, ΔG . The on- and off-frequency TMCs can be used to derive the IO function of the cochlear peak response to the on-frequency masker by plotting the off-frequency masker level at threshold for each masker-signal gap against the corresponding on-frequency threshold and correcting for the passive attenuation of the off-frequency masker response, P (Figure 3.1D). If the elicitor causes a gain reduction, ΔG , the IO functions for the with- and without-elicitor conditions (solid and dashed lines in Figure 3.1D) should differ by ΔG at low input levels, and converge towards higher levels. As a result, the with-elicitor IO function should grow less compressively (with a compression exponent of \tilde{c}).

Rather than, or in addition to, reducing cochlear gain, the elicitor might mask the signal directly through post-cochlear mechanisms. Direct masking by the elicitor would be equivalent to a reduction in signal level, which would cause the on- and off-frequency TMCs to shift rightwards (towards longer masker-signals gaps) by the same amount (Figure 3.1E). As a result, the off-frequency masker level at threshold would decrease equally at all masker-signal gaps and the amount of decrease would be equal to the amount of direct masking (compare solid and dashed blue lines in Figure 3.1E). In contrast, the on-frequency masker level at threshold would decrease in proportion to the slope of the on-frequency TMC; at short and long masker-signal gaps, the decrease would be equal to the decrease in the off-frequency masker level at threshold, but at intermediate gaps, the decrease would be greater by a factor of $1/c$. Thus, the effects of direct masking and gain reduction would be distinguishable in the on-frequency, but not in the off-frequency, masking condition. Importantly, direct masking by the elicitor should leave the derived IO function unchanged (Figure 3.1F).

The results of Micheyl and Collet (1996) suggest that the effect of a contralateral elicitor might depend on the order in which the with- and without-elicitor conditions are measured. They found a correlation between contralateral OAE suppression and elicitor-induced improvement in signal-in-noise detection when the with-elicitor conditions preceded the without-elicitor conditions, but not when the order was reversed. To control for any effects of condition order, the on-

frequency conditions with and without the elicitor were presented either separately, in different sessions, or interleaved within the same session.

3.2.2 METHODS

3.2.2.1 Participants

A total of 12 participants (7 males and 5 females, aged between 20-31 years) took part in this study. In six participants (L1-L6; 3 males and 3 females, aged between 20-26 years), on- and off-frequency TMCs both with and without the elicitor were measured. In the other six participants (L7-L12; 4 males and 2 females, aged between 20-31 years), a reduced set of conditions was measured, which excluded the off-frequency TMC with the elicitor. One participant (L2) took part in the piloting.

All participants were screened for normal hearing (absolute threshold < 20 dB HL) at audiometric frequencies between 0.25-6 kHz. They were also screened for normal middle-ear pressure (between -50-50 daPa) and normal middle-ear compliance (between 0.3-1.6 ml) using a GSI Tympanometer (Grason-Stadler, Eden Prairie, MN, USA). The participants reported no history of audiological or neurological disease and were not using any neuroactive medication. They were paid an inconvenience allowance. Informed written consent was obtained prior to participation. The experimental procedures were approved by the Ethics Committee of the Nottingham University School of Psychology and conformed to the guidelines of the Declaration of Helsinki at the time the

data were collected (version 6, 2008), but were not formally pre-registered online in accordance with the 2014 amendment to the declaration.

3.2.2.2 Experimental protocol and procedure

All thresholds were measured using a three-interval, three-alternative forced-choice adaptive tracking procedure. In the measurements of the signal detection threshold in quiet (needed to set the signal level for the TMC measurements), one interval, chosen randomly with equal *a priori* probability, contained the signal, and the other two contained silence. In the masker level at threshold (TMC) measurements, one interval contained the signal and masker and the other two contained the masker only. The trials were cued visually and separated by 500-ms gaps. The task was to select the signal interval by pressing the appropriate response button. Visual feedback was given after each trial indicating whether the participant had selected the correct or incorrect interval. The adaptive parameter was the signal level in the signal detection threshold measurements, and the masker level in the measurements of masker level at threshold. The signal level was varied according to a two-down, one-up procedure and the masker level according to a two-up, one-down, procedure, which tracks 70.7% correct performance (Levitt, 1971). The step size was 10 dB up to the first reversal, 5 dB up to the second reversal, and 2.5 dB for the remaining reversals. Each track was stopped after 12 reversals and lasted approximately 2 mins. The threshold was estimated as the average of the last 10 reversals within

each track. Six threshold estimates were acquired for each condition, and the set of three or more estimates with the least variance averaged to obtain a final threshold estimate. In the TMC measurements, different masker-signal gaps were measured in a random order. At least two hours of practice on the psychophysical task was given before data collection was started.

The on-frequency TMCs with and without the elicitor were measured either separately, in different sessions, or interleaved within the same session. In the interleaved sessions, the threshold tracks for the with- and without-elicitor conditions were alternated. Different sessions were conducted on different days and the order was counter-balanced across participants. The off-frequency conditions were measured in a separate session, with the with- and without-elicitor conditions interleaved. The off-frequency session was conducted before the on-frequency sessions in half of the participants, and after in the other half.

Each participant's MEMR threshold was measured for a broadband (0.125-4 kHz) noise with constant spectral density within its passband. The noise was presented to the same ear as the elicitor in the TMC measurements (left ear) and the reflex measured in the opposite (right) ear. The measurements were conducted with a GSI TympStar tympanometer. A reflex was taken as a change in middle-ear compliance of at least 0.02 ml.

3.2.2.3 Stimuli

The signal and maskers were sinusoids, presented to the right ear, and the elicitor was a broadband noise, presented to the left ear. The signal and on-frequency masker had a frequency of 2 kHz. The off-frequency masker had a frequency of 1.22 kHz, four auditory filter bandwidths (defined in ERBs; Glasberg and Moore, 1990) below the signal frequency. Results by Lopez-Poveda et al. (2003) suggest that, at four ERBs below the signal frequency, the tail response to the off-frequency masker is passive. The elicitor was bandpass-filtered to a range of 20 ERBs around the signal frequency (i.e., between 0.531-6.308 kHz). Within its passband, it was filtered to elicit equal energy per ERB (Glasberg and Moore, 2000). The filtering was conducted in the frequency domain using a 2^{19} -point fast Fourier transform (FFT) to create a 21.475-s cyclical noise buffer, which was played continuously throughout each threshold track. The bandpass filter was implemented as a boxcar. The signal and masker had durations of 5 and 25 ms, respectively, measured between the -3-dB points. All stimuli were gated on and off with quarter-sine and quarter-cosine ramps, respectively, with a duration of 5 ms (between 0 and 1). The masker-signal gap (measured between the -3-dB points) was varied between 5 and 30 ms in 5-ms steps. Not all masker-signal gaps were measured for all participants. The signal was presented at 10 dB above the signal detection threshold in quiet for each participant. The masker level was varied adaptively. The elicitor was presented at 40 dB SPL per ERB (corresponding to an overall level of 53.8 dB SPL). The

elicitor level was chosen to avoid eliciting the MEMR. The level corresponded to the lowest tympanometrically measured MEMR threshold across all participants, less 15 dB to account for the relative insensitivity of tympanometric MEMR threshold measurements (Goodman and Keefe, 2006).

All stimuli were generated digitally at a sampling rate of 24.414 kHz using TDT System 3 (Tucker-Davies Technologies, Alachua, FL, USA) and MATLAB (The Mathworks, Natick, MA, USA). They were digital-to-analogue converted with a 24-bit amplitude resolution (TDT RP2), amplified (TDT HB7), and presented through Sennheiser HD 600 headphones (Wedemark-Wennebostel, Germany) in a double-walled, sound-attenuating booth (IAC, Winchester, UK).

3.2.2.4 Statistical analysis

The data were analysed using linear mixed-effects regression models (LMMs), implemented in R (R Core Team, 2012). The models were constructed using the forward selection method. The model parameters were fitted using the *lme* function, which is part of the *nlme* package for R (Pinheiro J, 2014). Spearman correlations were calculated between the cochlear gain without the elicitor (G_{max}) and elicitor-induced change in cochlear gain (ΔG). The correlations were calculated using the *rcorr* function in R, which is part of the *Hmisc* package (Harrell et al., 2014).

3.2.3 RESULTS

The quiet detection threshold of the 5-ms, 2-kHz sinusoidal signal that was used for the TMC measurements was 23.5 ± 0.90 dB SPL, on average. The signal level was set at 10 dB above the individual signal detection thresholds. The MEMR threshold, measured tympanometrically, was 85 ± 3.1 dB SPL, on average, and the lowest threshold across participants was 70 dB SPL. Tympanometric measurements may overestimate the MEMR threshold by up to 15 dB (Goodman and Keefe, 2006). Therefore, the elicitor level was set to 40 dB SPL per ERB, which corresponds to an overall level of 53.8 dB SPL, that is, just below the lowest MEMR threshold across participants less 15 dB.

3.2.3.1 *Separate versus interleaved sessions*

The on-frequency masker levels at threshold with and without the elicitor were measured either in separate sessions on different days, or interleaved within the same session. However, there was no significant effect of session type, neither on the masker levels at threshold [tested with an LMM analysis, with elicitor condition (present/absent) and session type (separate/interleaved) as fixed factors, masker-signal gap as covariate, and participants as random intercepts; main effect of, and interactions with, session type: $\chi^2(1) = 1.042$, $p = 0.594$] nor on the differences in masker level at threshold for the with- and without-elicitor conditions [tested with an LMM with session type as fixed factor, masker-signal gap as covariate and participants as random intercepts: $\chi^2(1) =$

1.714, $p = 0.191$]. Therefore, the thresholds from the separate and interleaved sessions were averaged before further analysis.

3.2.3.2 Without-elicitor TMCs

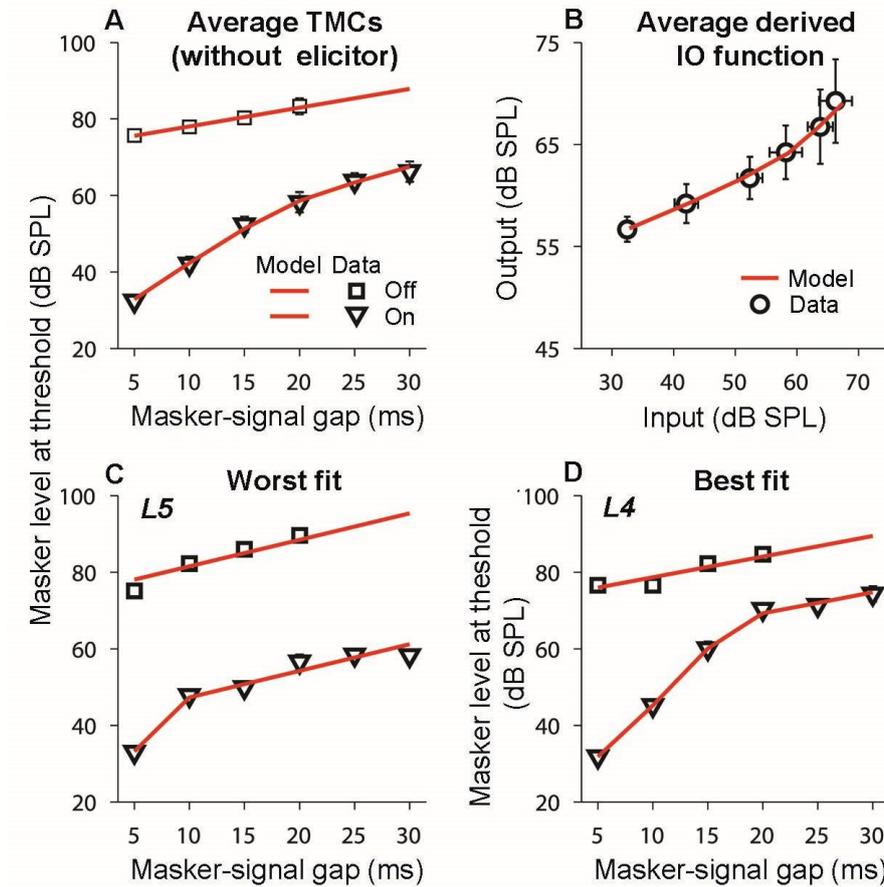


Figure 3.2: Panel A shows the measured (black) and fitted (red) on- and off-frequency TMCs without the elicitor present (open upside-down triangles and squares, respectively), averaged across all 12 participants. Panel B shows the IO functions derived from these data. Panels C and D show the measured and fitted data for the individuals for whom the model produced the worst and best fit. The error bars show the standard error of the mean (SEM). For all participants data, see Appendix A (Figure 1).

Figure 3.2A shows the on- and off-frequency TMCs without the elicitor, averaged across all 12 participants. The off-frequency masker level at threshold grew linearly with increasing masker-signal gap, as expected. The growth rate should correspond to the decay rate, μ , of the masker excitation over time. The slope of the on-frequency TMC was steeper at the shorter masker-signal gaps, and asymptoted towards the slope of the off-frequency TMC towards the longer gaps. The fact that the on-frequency TMC did not show a shallower slope at the shortest gaps measured indicates that the initial linear part of the cochlear IO function was not sampled (Figure 3.2B). An LMM analysis, with masking condition (on/off frequency) as fixed factor, masker-signal gap as covariate and participants as random intercepts, showed that the difference in slope between the on- and off-frequency TMCs was statistically significant [masking condition by masker-signal gap interaction: $\chi^2(2) = 420.538, p = < 0.001$].

Previously, the parameters of the cochlear IO function for the on-frequency masker (maximum gain, G_{max} , and compressive slope, c) were read out directly using selected data points from the TMCs (Nelson et al., 2001). Here, the parameters were derived by fitting a generic model of the signal and masker responses to the entire TMC dataset simultaneously. This makes the parameter estimates more robust to random measurement error. The signal and on-frequency masker peak responses were assumed to be equal to a piecewise linear function, f_a , of the relevant pressure level, L . f_a , was equal to the pressure level plus a

level-dependent gain, $G(L)$. In units of intensity, this becomes: $f_a(L) = 10^{(L+G(L))/10}$. $G(L)$ was maximal up to a first break point, BP_1 : $G(L \leq BP_1) = G_{max}$, and then decreased linearly, at a rate of $1 - c$, where c is the compression exponent, between BP_1 and a second break point, BP_2 :

$$G(L) = \begin{cases} G_{max}, & L < BP_1 \\ (c - 1)(L - BP_1) + G_{max}, & BP_1 \leq L < BP_2 \\ 0, & L \geq BP_2 \end{cases}$$

where $BP_2 = BP_1 - \frac{G_{max}}{c-1}$

The off-frequency masker tail response was assumed to be equal to a linear function, f_p , of the masker pressure level. f_p was equal to the pressure level minus a constant, P , representing the passive attenuation of the tail response: $f_p(L) = 10^{(L-P)/10}$. The masker responses (denoted E) were assumed to decay exponentially, at a rate μ , with increasing masker-signal gap, t : $E(t) = E(t = 0) \cdot e^{-\mu \cdot t}$. It was assumed that the masker level at threshold corresponded to a constant ratio, k , between the signal and masker responses. Using these assumptions, the on- and off-frequency masker levels at threshold, L_{on} and L_{off} , for each masker-signal gap, t , were predicted as:

$$L_{on}(t) = f_a^{-1} \left(f_a(L_s) / k \cdot e^{-\mu \cdot t} \right)$$

and $L_{\text{off}}(t) = f_p^{-1} \left(f_a(L_s) / k \cdot e^{-\mu \cdot t} \right)$, where f_a^{-1} and f_p^{-1} are the inverse of the functions f_a and f_p , and L_s is the signal sound pressure level. The sum of squared differences between all predicted and observed masker levels at threshold (on- and off-frequency) was minimised by varying the maximum gain, G_{max} , the compression exponent, c , the first break point, BP_1 , the passive off-frequency masker attenuation, P , the signal-to-masker ratio, k , and the masker decay rate, τ . The model was fitted to each participant separately.

	G_{max} (dB)	c	BP_1 (dB)	P (dB)
Mean	30.0	0.23	25.1	18.9
(±SE)	(± 1.24)	(± 0.026)	(± 1.10)	(± 2.21)

Table 3.1: Model estimates of G_{max} , c , BP_1 , and P , averaged across all 12 participants. For all individual model estimates see Appendix A (Table 1).

The model produced an excellent fit to the data (Figure 3.2). The root-mean-square deviation (RMSD) between the average predicted and observed masker levels at threshold was only 0.68 dB (Figure 3.2A, B). The individual RMSDs ranged from 0.71-2.60 dB; panels C and D in Figure 2 show the worst- and best-fit datasets, respectively. An LMM analysis, with masking condition (on/off frequency) and data type (predicted/observed) as fixed factors, masker-signal gap as covariate, and participants as random intercepts, showed that the predicted thresholds were not significantly different from the observed thresholds

[main effect of, and interactions with, data type: $\chi^2(2) = 0.084, p = 0.959$]. On average, the maximum gain, G_{max} , was estimated as 24.9 ± 2.56 dB, the compression exponent, c , as 0.27 ± 0.054 , and the passive attenuation, P , as 19.9 ± 2.71 dB (see Table 3.1 for the other model parameters). Across individuals, G_{max} ranged from 10.7 to 36.1 dB, and c ranged from 0.08 to 0.65 (see Figure 3.4A, B).

3.2.3.3 Effect of the elicitor

Panels A and B in Figure 3.3 show the average data from five of the first six participants (L1-L6), who measured both the on- and off-frequency masking conditions with the elicitor. One participant (L2) was omitted, because their off-frequency masker level at threshold for the longest masker-signal gap exceeded 100 dB SPL and was thus deemed unsafe to measure. Panels C and D show the data from the second six participants (L7-L12), who did not measure the off-frequency condition with the elicitor (see Section 3.2.2.1).

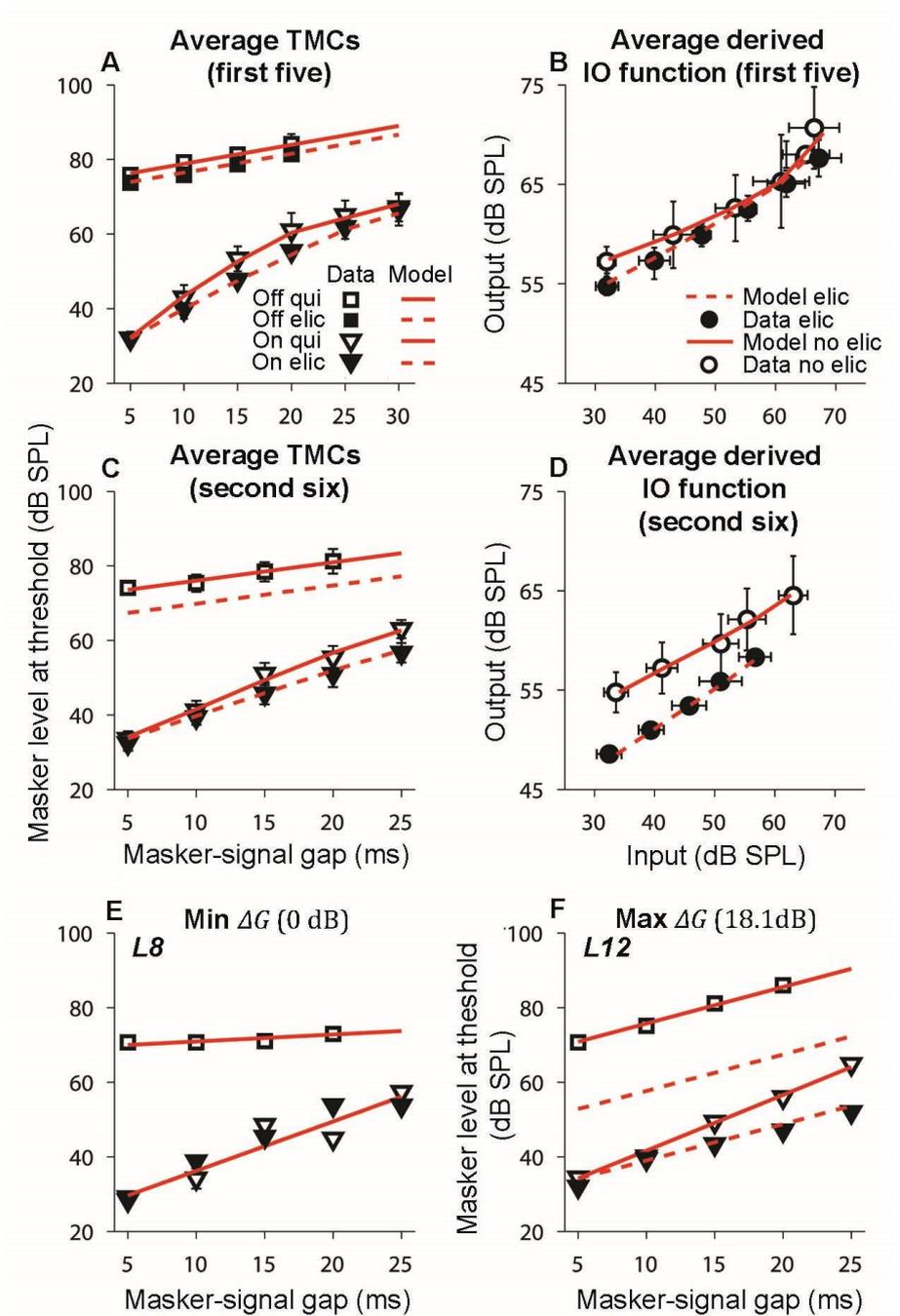


Figure 3.3: Panels A and C show the average TMCs for the first five (L1, L3-L6) and second six (L7-L12) participants, respectively. Measured (black) and fitted (red) TMCs are shown, both with (dashed lines and filled symbols) and without (solid lines and open symbols) the elicitor. The on- and off-frequency conditions are shown by upside-down triangles and

squares respectively. Panels B and D show the IO functions derived from these TMCs for each group. Panels E and F show the measured and fitted TMCs for the participants for whom the elicitor caused the minimum and maximum ΔG ; the minimum ΔG was 0 dB, so there was no difference in the fit with and without the elicitor. The error bars show the SEM.

Figure 3A shows that the elicitor caused the off-frequency masker level at threshold to decrease by about the same amount across all masker-signal gaps. This would be expected irrespective of whether the elicitor caused cochlear-gain reduction or direct, post-cochlear masking (see Figure 3.1C, E). The elicitor effect on the on-frequency masker level at threshold varied with the masker-signal gap (Figure 3.3A, C). At short gaps, there was little or no change in the on-frequency masker level at threshold. At intermediate gaps, the on-frequency masker level at threshold with and without the elicitor diverged and then converged again towards longer gaps. The derived IO functions with and without the elicitor showed a large difference at low input levels, and this difference appeared to decrease towards higher levels (Figure 3.3A). For both datasets, the IO function with the elicitor appeared to grow less compressively than that without the elicitor. This pattern of results is consistent with the idea that the elicitor caused a reduction in cochlear gain, but little or no direct masking. If the elicitor were reducing the amount of cochlear gain applied to the signal then the signal detection threshold in the presence of the elicitor would be expected to be higher than the signal detection threshold in quiet. However, no such increase

in the signal detection threshold in the presence of the elicitor was found [paired t -test: $t(5) = 1.26, p = 0.26$]. An LMM analysis of the data from all 12 participants, with elicitor condition (present/absent) and masking condition (on/off frequency) as fixed factors, masker-signal gap as covariate, and participants as random intercepts, showed that the elicitor effect was highly significant overall [main effect of, and interactions with, elicitor condition: $\chi^2(2) = 20.311, p < 0.001$]. Separate LMM analyses of the on- and off-frequency data (same factors, less masking condition) showed that the elicitor effect was significant for both conditions [on-frequency: $\chi^2(1) = 18.970, p < 0.001$; off-frequency: $\chi^2(1) = 6.746, p = 0.009$]. In the on-frequency condition, the elicitor caused a significant main effect [$\chi^2(1) = 16.353, p < 0.001$] as well as a marginal interaction with the masker-signal gap [$\chi^2(1) = 2.716, p = 0.099$]. In the off-frequency condition, the main effect was significant [$\chi^2(1) = 8.177, p = 0.004$], but the interaction was non-significant [$\chi^2(1) = 0.001, p = 0.980$].

The with-elicitor TMCs were fitted with the same model as was fitted to the without-elicitor TMCs. However, this time, it was assumed that G_{max} would be reduced by ΔG , where ΔG was a free parameter. All other parameters (i.e., the first and second break points, BP_1 and BP_2 , the passive off-frequency masker attenuation, P , the signal-to-masker ratio, k , and the masker decay rate, μ) were carried over from the without-elicitor fits. The fact that the break points were fixed meant that the compression exponent, c , changed according to: $\tilde{c} = 1 -$

$(G_{max} - \Delta G)/(BP_2 - BP_1)$. ΔG was fitted by minimizing the sum of squared differences between all predicted and observed with-elicitor thresholds, and the fitting was conducted separately for each participant.

Despite the fact that only one parameter was fitted, the model produced an excellent fit to the with-elicitor data. For the five participants who measured all conditions (L1, L3-L5; Figure 3.3A, B), the RMSD between the average predicted and observed with-elicitor thresholds was only 0.66 dB, and the individual RMSDs ranged from 2.09 to 3.45 dB. An LMM analysis, with masking condition (on/off frequency) and data type (predicted/observed) as fixed factors, masker-signal gap as covariate, and participants as random intercepts, showed that the predicted and observed thresholds were not significantly different from one another [$\chi^2(2) = 0.668, p = 0.716$]. For the second six participants, who did not measure the off-frequency condition with the elicitor (L7-L12; Figure 3.3C, D), the RMSD between the average predicted and observed with-elicitor thresholds was 0.79 dB, and the individual RMSDs ranged from 1.46 to 2.77 dB. An LMM analysis, with data type (predicted/observed) as fixed factor, masker-signal gap as covariate, and participants as random intercepts, showed again that the predicted and observed thresholds were not significantly different [$\chi^2(1) = 0.729, p = 0.393$]. For both groups, allowing G_{max} to be reduced yielded a significantly better fit to the with-elicitor data than fixing G_{max} at the value fitted to the without-elicitor thresholds [tested with an F -test; first five participants: $F(1,3) = 49.06, p = 0.006$; second six participants: $F(1,2) = 55.36, p =$

0.018]. On average across all 12 participants, the elicitor caused G_{max} to decrease by $\Delta G = 3.98 \pm 1.578$ dB. An unpaired t -test, assuming equal variance [confirmed with Levene's test for equality of variance: $F(1,9) = 3.21, p = 0.107$], showed that ΔG , was not significantly different across the two groups of participants [$t(9) = 0.97, p = 0.358$].

Across individuals, the elicitor-induced reduction in G_{max} , ΔG , showed a considerable degree of variability, ranging from 0 to 18.08 dB. As a result, the compression exponent with the elicitor, \tilde{c} , ranged from 0.106 to 1 (Figure 3.4C, D). There was no significant correlation between G_{max} without the elicitor and the elicitor-induced ΔG ($r = 0.19, p = 0.272$). Interestingly, there was also no correlation between G_{max} and the signal detection threshold in quiet ($r = -0.28, p = 0.378$), or between ΔG and the elicitor effect on the signal detection threshold in quiet ($r = 0.35, p = 0.499$).

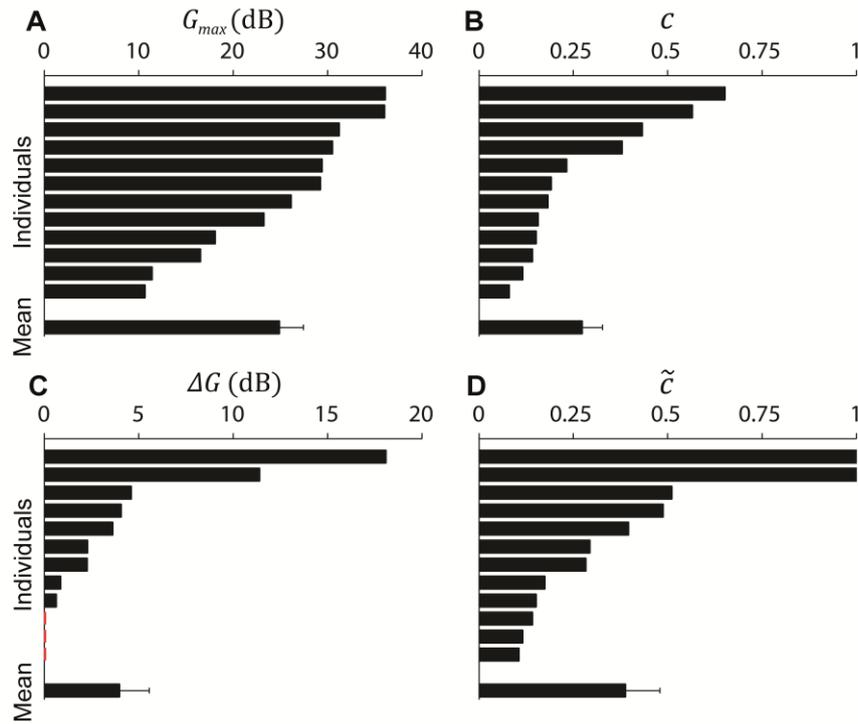


Figure 3.4: Individual model parameter estimates for the maximum cochlear gain (G_{max} , panel A), the compression exponent without the elicitor (c , panel B), the reduction in gain caused by the elicitor (ΔG panel C) and the compression exponent with the elicitor (\tilde{c} , panel D). For all parameters see Appendix A (Table 1).

3.3 EXPERIMENT 2: CONTRALATERAL SUPPRESSION OF OTOACOUSTIC EMISSIONS

3.3.1 GENERAL OUTLINE AND EXPECTATIONS

In this experiment, the effect of the contralateral MOCR elicitor used in Experiment 1 on OAEs evoked by an ipsilateral probe stimulus was

measured. The probe was a click in this case, rather than a tone pip as in Experiment 1. Typically, click-evoked OAEs are dominated by frequencies around 2 kHz, which corresponds to the signal frequency used in Experiment 1. Inspection of the current OAE spectra revealed that the same was true here. The probe was presented at a level of either 60 or 70 dB peak-equivalent (pe) SPL. Using two different probe levels makes it possible to measure the rate of compressive growth of the OAE amplitude with increasing probe level, which reflects the compressive growth of the cochlear response. Elicitation of the MOCR reduces cochlear gain and should thus suppress the amplitude of the probe-evoked OAEs. This effect is referred to as “amplitude suppression” and is the most commonly used measure of MOCR-induced OAE suppression (Guinan, 2010). MOCR elicitation also reduces the compressiveness of the cochlear response, and should thus also reduce the compressiveness in the growth of OAE amplitude with increasing probe level. This effect is referred to as “input-output (IO) suppression” (Veuille et al., 1996). It has been argued that IO suppression should be a more reliable measure of MOCR-induced reduction in cochlear gain, because it should be less affected by factors unrelated to cochlear gain. Here, both measures were used. Due to the non-linear nature of the mechanisms involved in the generation of OAEs, neither amplitude nor IO suppression of OAEs would be expected to represent a quantitative measure of MOCR-induced reduction in cochlear gain (Puria et al., 1996).

Experiment 2 was conducted after Experiment 1, using the same group of participants. As in Experiment 1, the conditions with and without the elicitor were measured either in separate sessions or interleaved within a single session in order to control for any effect of condition order. In this case, all sessions were conducted on the same day, with breaks between sessions.

3.3.2 METHODS

3.3.2.1 OAE measurements

Click-evoked OAEs were recorded using an in-house system (MLS 2001) consisting of a digital signal processing board controlled by custom-written software (Visual Basic). The clicks had a 100- μ s duration and were generated at a 30-kHz sampling rate. They were presented at a rate of 20/s using a general-purpose OAE transducer (Otodynamics, Hatfield, UK). The OAEs were recorded using the transducer microphone and digitized with a 30-kHz sampling rate and 18-bit amplitude resolution. They were averaged online over 2000 trials. Two such averages (referred to as “replicates”) were recorded for each click level (60 and 70 dB pe SPL) and elicitor condition (present/absent). Trials were rejected if the response amplitude exceeded 5 mPa within the period from 6-16 ms after the click. Each replicate took ~2 min to acquire, similar to the adaptive tracks in Experiment 1. The elicitor was presented continuously throughout the replicates for the with-elicitor condition. It was filtered in the same way, and presented at the same level and through the same

headphones (Sennheiser HD 600), as in Experiment 1. Within a session, replicates were measured contiguously. Different sessions were separated by breaks of at least 5 min. The OAE measurements were performed in the same sound-attenuating booth as Experiment 1. Participants watched a silent subtitled movie of their own choice to stay alert.

3.3.2.2 OAE data analysis

Offline-analysis of OAEs was performed in MATLAB. First, the OAEs were filtered between 250 Hz and 6 kHz by applying a 2nd-order Butterworth bandpass filter in both the forward and reverse time direction to create zero phase delay. To minimize the stimulus artefact, the OAEs were windowed between 6-16 ms after the click. The window edges were rounded according to 2-ms quarter-sine and -cosine functions. The OAE amplitude for each condition was taken as the integral of the co-spectrum between the respective replicates. The co-spectrum is the real part of the cross-spectrum (Marshall and Heller, 1996). OAEs were accepted as valid only if the correlation between the two replicates for each condition, referred to as “reproducibility”, was >0.7 . The reproducibility for all included OAEs averaged $0.95 (\pm 0.007)$.

The methods used for the statistical analysis of the OAE data were the same as those used in Experiment 1. In addition, Spearman correlations were calculated between the elicitor-induced OAE suppression (Experiment 2) and the elicitor-induced cochlear-gain reduction

estimated in Experiment 1. As before, the correlations were calculated using the *rcorr* function in R.

3.3.3 RESULTS

3.3.3.1 Separate versus interleaved sessions

OAE amplitude measurements with and without the elicitor were conducted either in separate sessions or interleaved within the same session. While there was no significant difference in the overall OAE amplitudes between these two types of session [tested with an LMM analysis with elicitor condition (present/absent), session type (separate/interleaved) and click level (60/70 dB pe SPL) as fixed factors, and participants as random intercepts; main effect of, and interactions with, session type: $\chi^2(4) = 1.358$, $p = 0.852$; Figure 3.5A], a significant difference between the separate and interleaved sessions was observed in the OAE suppression values [i.e., the differences in OAE amplitude between the with- and without-elicitor conditions; similar LMM analysis, less the elicitor condition factor: $\chi^2(2) = 7.542$, $p = 0.023$; Figure 3.5B]. Therefore, the OAE data were analysed separately for each session type.

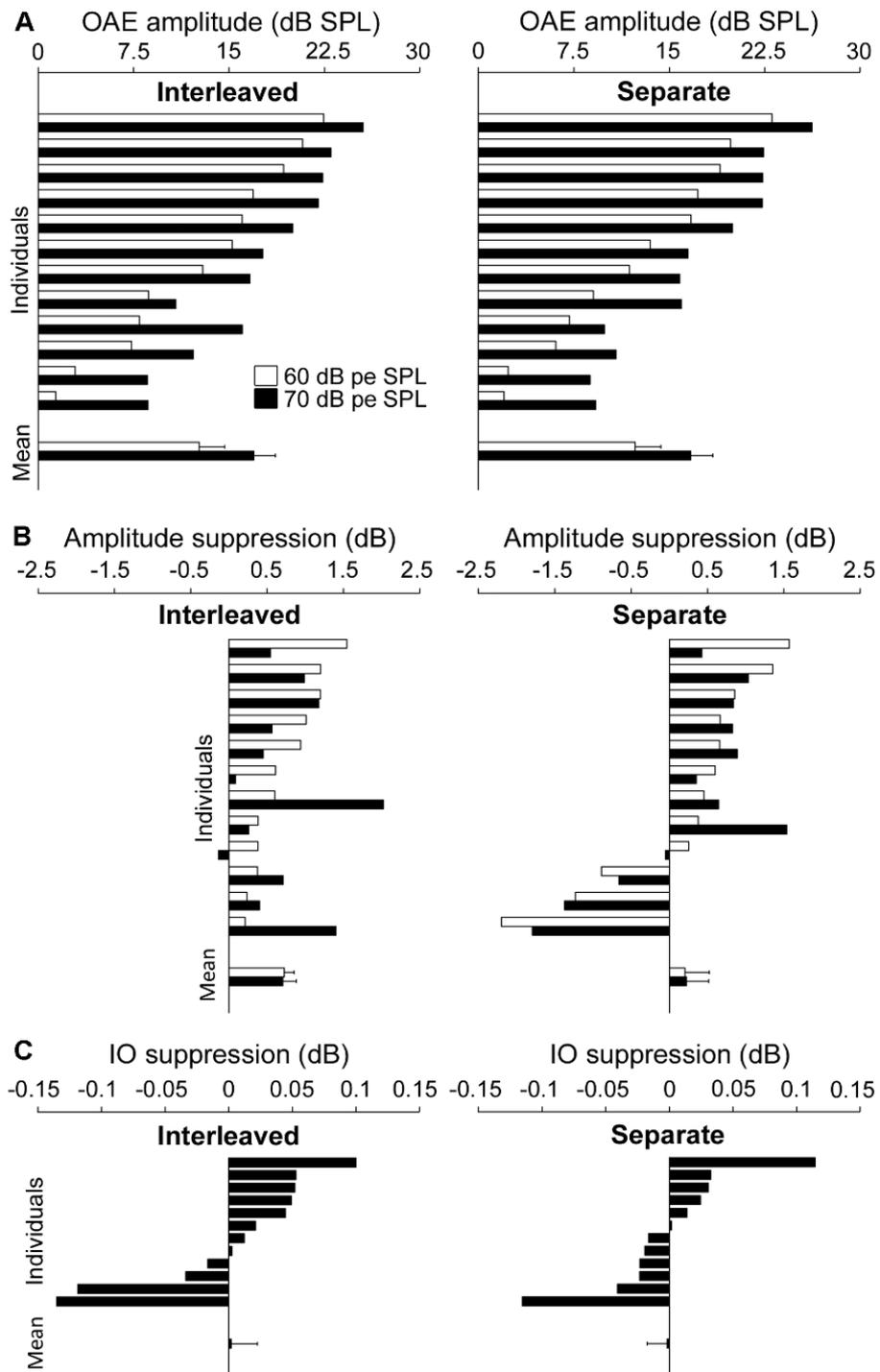


Figure 3.5: Panel A shows the OAE amplitudes without the elicitor for the interleaved (left) and separate (right) conditions. Panel B shows the OAE amplitude suppression by the elicitor for the interleaved (left) and separate (right) conditions. Individual and average data are shown for the 60- and

70-dB SPL clicks (white and black bars respectively). Panel C shows the individual and average OAE IO suppression by the elicitor for the interleaved (left) and separate (right) conditions. For the average data, error bars show the SEM.

3.3.3.2 OAE suppression

The OAE amplitudes were larger for the 70- than 60-dB pe SPL clicks (Figure 3.5A). This was true for both the separate and interleaved sessions [tested with separate LMM analyses, with elicitor condition and click level as fixed factors, and participants as random intercepts; main effect of click level: interleaved: $\chi^2(1) = 66.681$, $p = < 0.001$; separate: $\chi^2(1) = 64.356$, $p = < 0.001$]. Furthermore, the OAE amplitudes were smaller for the with- than without-elicitor conditions. However, the elicitor effect was significant only for the interleaved session [main effect of elicitor: $\chi^2(1) = 4.947$, $p = 0.026$], but not for the separate sessions [$\chi^2(1) = 0.425$, $p = 0.514$]. This was because, in the interleaved session (left panel in Figure 3.5B), the elicitor caused a reduction in OAE amplitude in all participants but one. In contrast, in the separate session (right panel), three of the 12 participants showed a large elicitor-related increase in OAE amplitude for both click levels. In the interleaved session, the OAE amplitude suppression averaged to 0.714 dB and ranged from -0.140 to 2.025 dB; in the separate session, the suppression averaged to 0.213 dB and ranged from -2.203 to 1.570 dB. In neither session did the elicitor effect significantly depend on the click level

[elicitor by click level interaction; interleaved: $\chi^2(1) = 0.001, p = 0.976$; separate: $\chi^2(1) = 0.001, p = 0.979$]. Thus, on average, neither session yielded any IO suppression (Figure 3.5C).

3.3.3.3 Correlations with psychophysical data

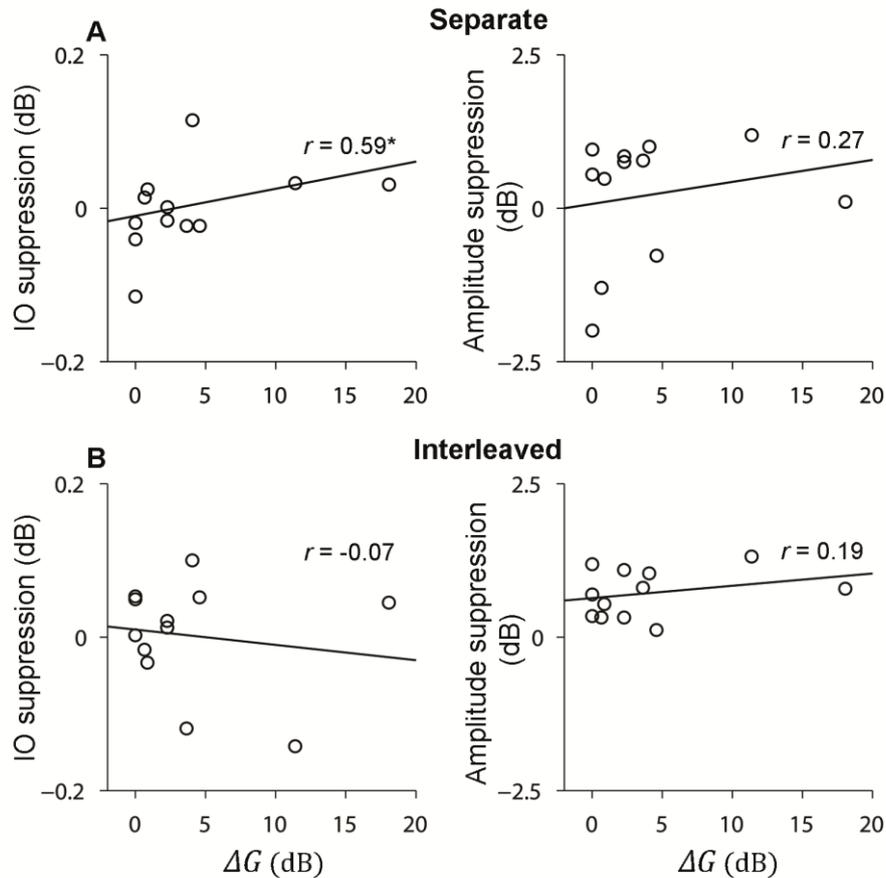


Figure 3.6: Correlations between OAE estimates and psychophysical estimates of cochlear-gain reduction by the elicitor. Psychophysical estimates of reduction in gain (ΔG) are plotted both against OAE IO suppression (left) and OAE amplitude suppression (right), for the separate (panel A) and interleaved (panel B) sessions. In each panel, the Spearman's rank correlation coefficient (r) and a linear regression fit (solid line) are shown.

Two-tailed Spearman correlations were calculated between the elicitor-induced cochlear-gain reduction, ΔG , estimated in Experiment 1 and the OAE amplitude and IO suppression measured in Experiment 2 (Figure 3.6). While there was a tendency for greater suppression for greater ΔG in most cases, only the correlation with the IO suppression measured in the separate sessions reached significance (separate: $r = 0.59$, $p = 0.043$; interleaved: $r = -0.07$, $p = 0.828$; Figure 3.6A).

3.4 DISCUSSION

This study was aimed at developing a psychophysical procedure to obtain a quantitative estimate of MOCR-induced reduction in cochlear gain in humans. For that, the established TMC method for estimating cochlear gain and cochlear compression (Nelson et al., 2001) was modified by shortening the masker stimulus so the masker itself would not be able to elicit the MOCR in time to affect the detectability of the signal. A new way of estimating cochlear gain and cochlear compression from TMC data was also developed, whereby the entire data set was fitted simultaneously with a generic model of the signal and masker responses, rather than being read out from selected data points (see Yasin et al., 2013b). The current method minimizes the effect of noise within data points and avoids the arbitrariness inherent in data point selection. Using this approach, the reduction in cochlear gain induced by a contralateral broadband-noise MOCR elicitor was estimated. It is shown that, when using a fixed signal level across the with- and without-

elicitor conditions, the on-frequency masking condition is essential for distinguishing between elicitor-induced reduction in cochlear gain and direct, post-cochlear masking; in the on-frequency condition, gain reduction and direct masking produce distinct patterns of effects, whereas in the off-frequency masking condition, the effects are indistinguishable.

The model, which was based on simple approximations of the active and passive cochlear IO functions, produced an excellent fit to both the with- and without-elicitor data. For the without-elicitor data, which were fitted first, the model coped well with the inter-individual variability in the TMCs, which was considerable despite all participants having audiometrically normal hearing. The estimated maximum cochlear gain without the elicitor, G_{max} , varied from as little as ~ 10 dB to as much as ~ 35 dB across participants, suggesting that, even when hearing is audiometrically normal, the integrity of the cochlear amplifier can vary substantially. This is consistent with previous results (e.g., Yasin et al., 2013). The average estimated maximum cochlear gain across all participants was ~ 25 dB. This is smaller, by ~ 25 dB, than estimates obtained in previous psychophysical studies (reviewed in Yasin et al., 2013b). The difference is due to the fact that the current study took account of the passive attenuation of the tail response to the off-frequency masker. Previous psychophysical studies have discounted this passive attenuation and, as a result, may have overestimated the maximum cochlear gain of the peak response to the on-frequency

masker. However, our estimates of maximum gain are slightly lower than has typically been found in squirrel monkeys (18-42 dB; Rhode, 1971, 1973, 1978, 1980), and considerably lower than those found in chinchillas (50-80 dB; Robles et al., 1986; Ruggero et al., 1997). It should be noted that the estimate of passive attenuation may have been considerably smaller had estimates been made using another method, such as that used by Oxenham and Plack (1997). It is possible that at least some of the difference between the on- and off-frequency masker thresholds at the longest masker-signal gaps, which has been consistently observed in previous studies using the TMC and FDMC method (e.g. Lopez-Poveda et al., 2003; Yasin et al., 2013), is due, not to passive attenuation, but to other, perhaps attentional, factors. It is important that future studies clarify the contribution of attentional factors to psychophysical estimates of cochlear gain (see Chapter 5 for a discussion of the possible attentional effects that may occur, and how future studies may be able to test for them). The average estimated compression exponent for the current without-elicitor data was 0.27, which is similar to previous psychophysical (Yasin et al., 2014) and physiological estimates (for review, see Robles and Ruggero, 2001).

The model also managed to reproduce the different pattern of effects of the elicitor in the on- and off-frequency TMCs. The estimated elicitor-induced reduction in maximum cochlear gain was ~4 dB, on average, and ranged from 0 to as much as ~18 dB across participants. In half of the participants, the elicitor effect was measured only in the on-frequency

condition. Despite this, the estimated gain reduction did not differ significantly between the two groups. This suggests that the off-frequency condition is not essential for estimating MOCR-induced cochlear-gain reduction.

Like the current study, Aguilar et al. (2013) measured cochlear-gain reduction by a contralateral noise elicitor using the TMC method, although they use a 4-kHz rather than 2-kHz signal frequency and only measured off-frequency TMCs. Despite presenting the elicitor at a higher level (60 vs. 53.8 dB SPL), Aguilar et al. found no significant elicitor-induced gain reduction. This was probably due to the longer masker duration used by Aguilar et al. (200 vs. 25 ms long), which means that the masker itself may have elicited the MOCR in time to affect the cochlear gain of the signal, and thus obscured any additional effect by the elicitor.

The current elicitor effect (~ 4 dB) was considerably smaller than the effect found by Krull and Strickland (2008) and Roverud and Strickland (2010). Both Krull and Strickland and Roverud and Strickland used a sinusoidal elicitor at the signal frequency, presented ipsilateral to the signal and masker. Given that, like Aguilar et al. (2013), they both measured only the off-frequency TMCs, it cannot be excluded that their larger elicitor effects reflect direct, post-cochlear masking of the signal by the elicitor, rather than gain reduction. Previous results from OAE suppression measurements suggests that a sinusoidal elicitor should be

less effective at inducing a gain reduction than a broadband-noise elicitor, as used in the current study (Lilaonitkul and Guinan, 2009a, b).

Yasin et al. (2014) used an ipsilateral noise elicitor, but with a narrower bandwidth than either the current, or Aguilar et al. (2013)'s, elicitor. Unlike the previous studies, they used varying elicitor levels and varying gaps between the elicitor and masker. The largest elicitor effects were found when the elicitor and masker were presented with a 0-ms gap. In this condition, an elicitor level of only 40 dB SPL produced an average gain reduction effect of ~16 dB, four times larger than the ~4-dB effect found in the current study with a 53.8-dB SPL elicitor level. This is surprising, because OAE suppression data from humans and anatomical data from monkeys suggest that, in primates, the crossed and uncrossed medial olivocochlear efferents are approximately equally strong (Bodian and Gucer, 1980; Thompson and Thompson, 1986; Lilaonitkul and Guinan, 2009a). Thus, Yasin et al. (2014)'s elicitor effect may have been expected to be comparable to, or even smaller than, that found in the current study. Given that they used an ipsilateral elicitor, it is possible that their elicitor effect was caused by nonlinear interactions due to direct temporal overlap between the elicitor and masker responses. Nonlinear temporal interaction is thought to underlie the “temporal suppression” effect in OAEs and auditory brainstem responses (Kapadia and Lutman, 2000a, b; Harte et al., 2005; Bianchi et al., 2013), whereby a closely preceding “suppressor” sound decreases the estimated cochlear response to a probe sound.

In the current study, the elicitor had a significant effect on both the on- and off-frequency TMCs, but did not significantly increase the signal detection threshold in quiet. Moreover, there was no significant correlation between the elicitor-induced gain reduction, ΔG , and the elicitor effect on the signal quiet threshold across participants. This surprising finding may be related to the commonplace observation that absolute hearing thresholds do not correlate well with measures of OHC integrity, such as auditory frequency selectivity (Tyler, 1986; Moore et al., 1999) and cochlear compression (Plack et al., 2004). Consistent with this, the current study found no correlation between the signal quiet threshold and the maximum cochlear gain, G_{max} , without the elicitor. This may have occurred because at least some of the internal noise that determines the absolute threshold might occur at or before the stage at which cochlear amplification occurs (Gebeshuber, 2000). Any pre-amplification noise would diminish the influence of cochlear gain on the signal-to-noise ratio at absolute threshold.

The large variation in maximum cochlear gain (G_{max}) across participants in the current study, suggests that some participants may be suffering from an “obscure auditory dysfunction” (reviewed in Plack et al., 2014). Obscure auditory dysfunction refers to the fact that some listeners with audiometrically normal hearing report difficulties understanding speech in noisy environments. Obscure auditory dysfunction is thought to be due to selective loss of high-threshold auditory nerve fibres caused by noise exposure (Kujawa and Liberman, 2009) or ageing (Sergeyenko et

al., 2013). The current results suggest that OHC damage may also be a contributing factor to obscure auditory dysfunction.

There was no correlation between the maximum cochlear gain, G_{max} , and the amount of gain reduction by the elicitor, ΔG . This was probably because, firstly, all participants in the current study showed substantial cochlear gain (25 dB, on average) and, secondly, the elicitor used was relatively low in level (to avoid eliciting the MEMR) and thus did not cause a large reduction in gain (4 dB, on average). If a more varied set of participants had been used and a higher-level elicitor, a significant correlation may have been found, because the amount of gain reduction would have been limited by the baseline amount of gain without the elicitor (Plack et al., 2004).

Animal work suggests that the MOCR protects the auditory system from noise-induced hearing loss (Maison and Liberman, 2000). This raises the exciting possibility that the quantitative measure of MOCR-induced cochlear-gain reduction developed in the current study could be used as a predictor of whether or not a given individual is likely to develop a hearing loss in later life. A predictive measure of susceptibility to hearing loss would help to preserve healthy hearing by influencing lifestyle choices and encouraging preventative behaviours.

Unlike the results from psychophysical measurements, the results from the OAE suppression measurements were sensitive to the order in which the with- and without-elicitor conditions were measured; only when the

with- and without-elicitor conditions were interleaved within the same session did the elicitor cause a significant suppression in OAE amplitude. The elicitor effect in the interleaved session supports the conclusion from the psychophysical results that the elicitor caused a reduction in cochlear gain through activating the uncrossed MOCR. When the with- and without-elicitor conditions were measured in separate sessions, three of the participants showed a large elicitor-induced enhancement in OAE amplitude. In these three participants, the without-elicitor session preceded the with-elicitor session. The order effect found in the current study might be related to that found by Micheyl and Collet (1996). They measured correlation between OAE amplitude suppression and improvement in signal-in-noise audibility by a contralateral elicitor and found significance only when the with-elicitor condition preceded the without-elicitor condition. The fact that, in the current study, the order effect was present in the OAE, but not the psychophysical, data suggests that the effect is specific to OAEs and thus not related to MOCR-induced reduction in cochlear gain. The effect may thus represent a confound when measuring MOCR-induced gain reduction using only OAEs.

While there was significant OAE amplitude suppression in the interleaved session, there was no significant IO suppression for either session type. However, this was not because most participants showed IO suppression around zero, but rather, because some participants showed large positive, and others, large negative IO suppression. In the participants who showed positive IO suppression, the amount of

suppression was comparable to that found in previous studies (de Boer and Thornton, 2007; de Boer and Thornton, 2008; de Boer et al., 2012). This was true for both the interleaved and separate sessions. The finding of negative IO suppression was unexpected, although such an effect has also been found previously (de Boer et al., 2012).

The elicitor effect on the OAE amplitudes and psychophysical TMCs would be assumed to reflect the same underlying processes, namely activation of the MOCR and the resultant reduction in cochlear gain. Therefore, OAE-based and psychophysical estimates of gain reduction would be expected to be correlated. The only significant correlation found between the elicitor effect on psychophysical estimates of cochlear gain and OAE suppression estimates was for OAE IO suppression measured in the separate sessions, and this correlation was quite weak. The absence of a strong correlation may be due to the fact that OAE amplitudes are affected by many factors other than cochlear gain, such as the degree and distribution of cochlear irregularities (Shera and Guinan, 1999) and the transmission properties of the middle ear (Probst et al., 1991). Furthermore, it has been suggested that the MOCR is susceptible to attentional modulation (Giraud et al., 1995; Maison et al., 2001; de Boer and Thornton, 2007; see Chapter 1, Section 1.2.3). Thus, given that the OAE suppression measurements were conducted under passive listening conditions, differences in attentional state may also have contributed to the differences between the psychophysical and OAE results. Finally, evidence has been reported suggesting that the MOCR is

influenced by auditory training (de Boer and Thornton, 2008). Thus, the relationship between the psychophysical and OAE results may have been different had the OAEs been measured before, rather than after, the psychophysical experiment.

In the current experiments, the elicitor was set at a conservative level to ensure that it would not activate MEMR. It could be argued that this was unnecessary, because, whilst the MEMR can be activated by higher frequencies, the reflex causes little attenuation above ~ 1 kHz (Nuttall, 1974). However, whilst, in the psychophysical experiment, activation of the MEMR might not have influenced the 2-kHz signal and masker, it may well have affected the off-frequency masker and thus changed the relationship between the two masking conditions. Using a higher signal frequency (e.g., 4 kHz) would be expected to mitigate this problem. In the current study, the signal was placed near the spectral peak of the click-evoked OAE, where the contralateral OAE suppression effect is easiest to detect. At higher frequencies, contralateral suppression of OAEs has been shown to be weak (Lilaonitkul and Guinan, 2009a).

3.5 CONCLUSION

In the current study, a method for obtaining a quantitative estimate of cochlear-gain reduction by the contralateral MOCR in humans was refined. The method is based on the psychophysical TMC method for estimating cochlear gain and compression proposed by Nelson et al.

(2001). Despite the fact that the contralateral elicitor was presented at a relatively low level, it had a highly significant effect on both the on- and off-frequency TMCs. The pattern of these effects conformed remarkably well with predictions based on widely accepted model approximations of the active and passive cochlear IO functions. The new method seems preferable to the classical OAE-based measurements of MOCR-induced gain reduction, because it is less affected by factors unrelated to cochlear gain and can be used to measure MOCR effects at higher frequencies, which would be less affected by the MEMR.

CHAPTER 4

CAN THE IPSILATERAL MEDIAL OLIVOCOCHLEAR REFLEX BE MEASURED QUANTITATIVELY USING THE FIXED- DURATION MASKING CURVE METHOD?

4.1 INTRODUCTION

In a recent study, Yasin et al. (2014) estimated that a low-level ipsilateral elicitor caused an MOCR-induced cochlear-gain reduction that was four times larger than that found in Chapter 3 with a contralateral elicitor. In Chapter 3, the elicitor was a broadband noise (> 3.5 octaves wide) presented at 53.8 dB SPL, whereas in Yasin et al. (2014), the elicitor was

a narrowband noise (< 0.5 octaves wide) presented at 40 dB SPL. The findings of Yasin et al. (2014) are surprising given recent OAE suppression measurements, which suggest that the largest gain reductions are produced when the elicitor is broadband, with the effect decreasing markedly with decreasing bandwidth (Lilaonitkul and Guinan, 2009a). Moreover, for broadband elicitors (>~2 octaves wide), the MOCR effect was found to be of a similar magnitude irrespective of whether the elicitor was presented ipsilateral or contralateral to the probe. The elicitor used by Yasin et al. (2014) would therefore have been expected to produce a smaller effect than the one used in Chapter 3. The aim of the current study was to re-examine Yasin et al. (2014)'s surprising finding.

Yasin et al. (2014) presented their ipsilateral elicitor before the masker and signal. They used various different elicitor levels and temporal gaps between the elicitor and masker. The largest elicitor effects were found for a gap of 0-ms. At the 50-ms gap, the effect had decayed substantially. In the 0-ms gap condition, the lowest elicitor level that caused a significant effect was 40 dB SPL. Yasin et al. (2014) interpreted the effect as MOCR-induced cochlear-gain reduction. However, it is possible that, for a gap of 0 ms, the elicitor effect was, at least partly, caused by direct nonlinear interactions due to partial temporal overlap between the cochlear responses to the elicitor and masker. Such a “temporal suppression” effect was first observed in OAE measurements, where it was found that the OAE amplitude in response to a probe click was

reduced by a preceding click (Kemp and Chum, 1980; Kapadia and Lutman, 2000a, b). This effect has also been measured in auditory brainstem responses and there is evidence suggesting that it might contribute to the precedence effect (Bianchi et al., 2013). The precedence effect describes the phenomena whereby two sounds that are close together in time can become fused into a single auditory image the perceived location of which is strongly biased towards that of the leading sound.

To estimate cochlear-gain reduction, Yasin et al. (2014) used a modified version of the TMC method (see Section 3.2.1), which they have called the “fixed-duration masking curve” (FDMC) method (see, Yasin et al., 2013b, a). In the FDMC method, the signal is made more or less detectable by changing the relative duration of the signal and masker, rather than by changing the masker-signal gap as in the TMC method. The main advantage of the FDMC method is that the time from the masker onset to the signal offset remains fixed. This removes the risk, present at large masker-signal gaps in the TMC method, that the masker will itself elicit the MOCR and cause a reduction in the amount of cochlear gain applied to the signal. Such a reduction in the signal gain by the masker could lead to underestimation of cochlear gain, and thus underestimation of elicitor-induced gain reduction. However, the TMC method has produced similar estimates of cochlear gain to the FDMC method and lower compression estimates (the opposite to what would be expected if gain

was being underestimated at the longer masker-signal gaps in the TMC method; Yasin et al., 2013b).

In the current study, Yasin et al. (2014)'s findings were first replicated by using the FDMC method to estimate the amount of cochlear-gain reduction caused by a long-duration elicitor ("ipsi long"), presented ipsilateral to the signal and masker. As in Yasin et al. (2014), the signal level was adjusted to control for any masking of the signal by the elicitor. The elicitor had a similar bandwidth and level to the one used by Yasin et al. (2014). To test whether the effect of this elicitor was really caused by MOCR-induced cochlear-gain reduction, the effect of the ipsi-long elicitor was compared to the effect of an elicitor that produced the same amount of masking of the signal, but was too short to activate the MOCR in time to affect the masker. This elicitor will be referred to as "ipsi short". It was expected that, if the effect of the ipsi-long elicitor was only due to MOCR-induced cochlear-gain reduction, the ipsi-short elicitor would have no effect on the masker level at threshold. However, if the effect was due to other factors, such as temporal suppression, then the effect of the ipsi-short elicitor should be similar to that of the ipsi-long elicitor. The effect of the long-duration elicitor was also measured when it was presented contralateral to the signal and the masker ("contra long"), where it would be expected to elicit the MOCR but not cause any temporal suppression.

Cochlear-gain reduction caused by these different elicitors was estimated both psychophysically and using OAEs. This allowed whether or not an MOCR-induced cochlear-gain change had been elicited by any of the elicitors to be assessed independently. Typically, in previous OAE studies, MOCR-induced reduction in cochlear gain has been elicited using a continuous noise elicitor, presented contralateral to the probe (see Guinan, 2010, for review). In order to compare the effects of the ipsi-long, ipsi-short, and contra-long elicitors to these previous OAE suppression measurements, OAE suppression was also measured for a contralateral elicitor that was presented simultaneously with the click (“contra sim”). The other elicitors (ipsi long, ipsi short, and contra long) were presented before the probe as in the psychophysical measurements.

4.2 EXPERIMENT 1: FIXED-DURATION MASKING CURVE (FDMC) MEASUREMENTS

4.2.1 GENERAL OUTLINE AND EXPECTATIONS

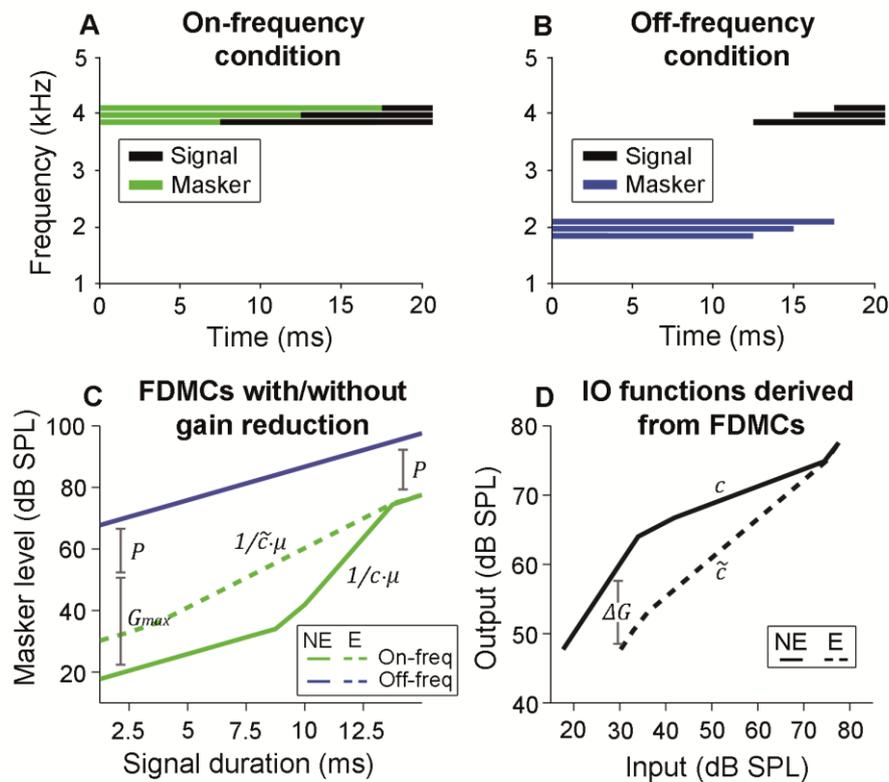


Figure 4.1: Panels A and B show a schematic representation of the spectral and temporal characteristics of the stimuli for the on- and off-frequency conditions. The signal and masker are represented by different colours (see legend). The stacked lines show the different relative durations of the signal and masker. Panel C shows simulated FDMCs for the on- and off-frequency conditions (green and blue, respectively) both without (solid lines) and with (dotted lines) the elicitor (labelled NE and E respectively), under the assumption that the elicitor causes only a reduction in cochlear

gain. Cochlear-gain reduction would be expected to have no effect on the off-frequency condition, so no dotted line is visible for this condition. In panel C, it is shown that the difference between the on- and off-frequency masker level at threshold is due, firstly, to the passive attenuation of the basilar membrane response to the off-frequency masker at the characteristic place of the signal (P), and, secondly, to the amount of cochlear gain applied to the on-frequency masker response (G_{\max}). The maximum rate of growth of the on-frequency FDMC without the elicitor is equal to $1/c \cdot \mu$, where c is the compression exponent without the elicitor and μ is the rate of decrease in masker effectiveness with decreasing masker duration (i.e., the slope of the off-frequency FDMC). The maximum growth rate with the elicitor is equal to $1/\tilde{c} \cdot \mu$, where \tilde{c} is the compression exponent with the elicitor present. Under the assumption that the elicitor reduces cochlear compression, $\tilde{c} > c$. Panel D shows the IO functions derived from the FDMCs in panel C. For the IO function without the elicitor, the rate of growth of the output at intermediate input levels is equal to c , which is the compression exponent. The difference in output between the with- and without-elicitor conditions is equal to the amount of gain reduction (ΔG).

In this experiment, FDMCs were measured for a 4-kHz sinusoidal signal, presented at 10 dB SL (Figure 4.1A, B). The masker was a 1-ERB-wide noise that was centred either at (Figure 4.1A) or well below (Figure 4.1B) the signal frequency (“on-” and “off-frequency” conditions, respectively). The signal and masker were presented to the right ear. The masker level

at threshold (i.e., the lowest masker level needed to mask the signal) was measured as a function of the masker and signal durations, with the total duration fixed at 20 ms. For the on-frequency masker, signal durations of 2.5, 7.5 and 12.5 ms were used (the corresponding masker durations were 17.5, 12.5, and 7.5 ms, respectively), so that cochlear gain could be estimated for a range of different masker levels. For the off-frequency masker, signal durations of 2.5, 5 and 7.5 ms (masker durations of 17.5, 15, and 12.5 ms) were used because piloting had shown that, for signal durations longer than 7.5 ms, the masker level at threshold would likely exceed safe-listening limits.

Unlike Yasin et al. (2014), who used tonal maskers, in the current study, narrowband-noise maskers, which produce stronger masking for short signals, were used. Narrowband noise maskers create more masking because fluctuations in their amplitude spectrum (which are not present for tonal maskers) can be confused with the signal (Neff, 1986; see section 4.4 for detailed discussion). This was so that cochlear-gain estimates could be made at low on-frequency masker levels, at which cochlear gain and MOCR-induced cochlear-gain reduction would be expected to be maximal. Pilot measurements showed that, even for the shortest signal duration, a tonal on-frequency masker produced less masking than in Yasin et al. (2014) and so the masker level at threshold was higher. The reason for this discrepancy could not be established.

FDMCs were measured either without an elicitor, or with one of three different noise elicitors presented just before the masker and signal. The ipsi-long and contra-long elicitors both had a duration of 500 ms and the ipsi-short elicitor had a duration of 15 ms. The ipsi-short elicitor was expected to be too short to elicit the MOCR in time to cause a reduction in the amount of cochlear gain applied to the masker (James et al., 2002a; Backus and Guinan, 2006).

As in Yasin et al. (2014), the signal was set to 10 dB above its detection threshold in quiet when no elicitor was present. When an elicitor was present, the signal was set to 10 dB above its masked threshold in order to compensate for any direct, post-cochlear masking of the signal by the elicitor. The ipsi-long and contra-long elicitors were both presented at 36.2 dB SPL/ERB (40 dB SPL overall level) to match the lowest elicitor level that caused a significant effect in Yasin et al. (2014). The level of the ipsi-short elicitor was set individually so that it would produce the same amount of masking as the ipsi-long elicitor. To determine this level, the signal level was set to its detection threshold in the presence of the ipsi-long elicitor and the ipsi-short elicitor level was adjusted until it just masked the signal at this level.

As in Chapter 3, an established model of the cochlear IO function was used to derive expectations about how the elicitor would affect the on- and off-frequency FDMCs (see Section 4.2.2 and Figure 4.1). In the FDMC method, the signal level is set so as to compensate for any direct, post-

cochlear masking by the elicitor, and, so, no effect of direct masking was expected. The FDMC method relies on the assumption that the decay rate of the masker response is independent of the masker frequency. As in the TMC method, the off-frequency masker level at threshold should increase linearly with increasing signal duration, and the rate of increase should correspond to the rate of decrease of the masker effectiveness with decreasing masker duration (solid blue line in Figure 4.1C). In contrast, the on-frequency masker response is active. As in the TMC method, the on-frequency masker level at threshold (solid green line in Figure 4.1C) will grow at the same rate as the off-frequency masker level at threshold at short and long signal durations. At intermediate durations, however, the rate of growth of the on-frequency masker level at threshold will be faster than that of the off-frequency threshold, by a factor corresponding to the reciprocal of the compression slope of the IO function, $1/c$ (see Chapter 3, Section 3.2.1). At short signal durations, the difference between the on- and off-frequency masker levels at threshold would be assumed to reflect the sum of the active amplification of the on-frequency masker response (G_{max}) and the passive attenuation of the off-frequency masker response at the signal frequency (P ; see Figure 4.1C). Towards longer signal durations, the difference decreases to the passive attenuation only.

If the elicitor causes a reduction in cochlear gain, no effect would be expected on the off-frequency masker level at threshold. This is because the tail of the off-frequency masker response would not be affected by

cochlear-gain reduction, and, although the signal response would be affected, any reduction in signal gain would have been compensated for by setting the signal at 10 dB above its masked threshold in the presence of the elicitor (for this reason no dotted line is visible for the off-frequency masking condition in Figure 4.1C). For the on-frequency masking condition, the effect of a gain reduction by the elicitor would be expected to depend on the signal duration. Generally, the gain reduction would affect the masker but not the signal, because the signal level is set to compensate for any gain reduction. The elicitor-induced increase in masker level at threshold should thus always correspond to the amount of cochlear-gain reduction caused by the elicitor. At short signal durations, the masker level is low and the cochlear gain applied to the masker will thus be large. As a result, the reduction in gain should also be large (compare solid and dashed green lines in Figure 4.1C). At intermediate signal durations, the gain reduction should decrease in proportion to the decrease in cochlear gain applied to the masker. The difference in the growth rate of masker level at threshold between the with- and without-elicitor conditions should correspond to the difference between the reciprocals of the compression slopes of the respective IO functions ($1/c - 1/\tilde{c}$; see Figure 1C). At long signal durations, the on-frequency masker response will become passive, like the off-frequency masker response, and so, no difference between the on-frequency masker levels at threshold for the with- and without-elicitor conditions would be expected.

As in the TMC method, the cochlear IO function of the on-frequency masker can be derived by plotting the off-frequency masker level at threshold for each signal duration against the corresponding on-frequency threshold and correcting for the passive attenuation of the off-frequency masker response, P (Figure 4.1D). If the elicitor causes a gain reduction, the masker IO functions with and without the elicitor (solid and dashed lines in Figure 4.1D) should differ by the amount of gain reduction, ΔG , at low input levels, and progressively less towards higher levels (Figure 4.1D). As a result, the masker IO function with the elicitor should grow less compressively than that without the elicitor (compare compression exponents c and \tilde{c} in Figure 4.1D).

4.2.2 METHODS

4.2.2.1 Participants

Five participants (2 males and 3 females, aged between 20-25 years) took part in this study. The same participants took part in the psychophysical and OAE experiments. The participants were screened for normal hearing (absolute threshold < 20 dB HL) at audiometric frequencies between 0.25-6 kHz. They were also screened for normal middle-ear pressure (between -50 - 50 daPa) and normal middle-ear compliance (between 0.3-1.6 ml) using a GSI Tympanometer (Grason-Stadler, Eden Prairie, MN, USA). None of the participants reported any history of audiological or neurological disease or were using neuroactive medication. They were paid an inconvenience

allowance. Informed written consent was obtained prior to participation. The experimental procedures were approved by the Ethics Committee of the Nottingham University School of Psychology and conformed to the guidelines of the Declaration of Helsinki at the time the data were collected (version 6, 2008). They were not formally pre-registered online in accordance with the 2014 amendment to the declaration.

4.2.2.2 Experimental protocol and procedure

All thresholds were measured using a three-interval, three-alternative forced-choice adaptive tracking procedure. Only one of the three intervals, chosen randomly with equal *a priori* probability, contained the signal. The trials were cued visually and separated by 500-ms gaps. The task was to select the signal interval by pressing the appropriate response button. Visual feedback was given after each trial indicating whether the participant had selected the correct or incorrect interval. In the measurements of the signal detection threshold in quiet and with the elicitors (needed to set the signal level for the FDMC measurements), the adaptive parameter was the signal level. In the measurements of masked thresholds (i.e., the masker level needed to just mask the signal), the masker level was the adaptive parameter. The signal level was varied according to a two-down, one-up, and the masker level according to a two-up, one-down, procedure, both of which track 70.7%-correct performance (Levitt, 1971). The step size was 10 dB up to the first reversal in the adaptive parameter, 5 dB up to the second reversal, and 2.5 dB for the remaining reversals. Each track was stopped after 10

reversals and lasted approximately 2 mins. The threshold was estimated as the average of the last 6 reversals within each track. Six threshold estimates were acquired for each condition and averaged. In the FDMC measurements, different signal durations and elicitor conditions were measured in a random order. At least two hours of practice on the psychophysical task were given before data collection was started.

Each participant's MEMR threshold was measured for a broadband (0.125-4 kHz) noise with constant spectral density within its passband. The reflex was measured in the right ear and the noise was presented either ipsilateral or contralateral to the measurement ear. The measurements were conducted with a GSI Tympanometer. A reflex was taken as a change in middle-ear compliance of at least 0.02 ml.

4.2.2.3 Stimuli

The signal was a sinusoid and the masker was a narrowband noise, presented to the right ear. The signal was at 4 kHz. The masker was a 1-ERB-wide noise that was centred either at 4 kHz (on frequency) or at 2 kHz (off frequency). Results by Lopez-Poveda et al. (2003) suggest that the response to this off-frequency masker would be completely passive. The elicitors were bandpass-filtered to a range of 2.4 ERBs around the signal frequency (i.e., between 3500-4500 Hz). Within its passband, the elicitor was filtered to elicit equal energy per ERB (Glasberg and Moore, 2000). The filtering was conducted in the frequency domain using a 2^{19} -point FFT to create a 21.475-s cyclical noise buffer, which was played

continuously throughout each threshold track. The bandpass filter was implemented as a boxcar. For the on-frequency masker, signal durations of 2.5, 7.5 and 12.5 ms were used. For the off-frequency masker, signal durations of 2.5, 5 and 7.5 ms were used. The corresponding masker durations were chosen so that the total time from the masker onset to signal offset was 20 ms. The signal and masker durations are measured between the -3 -dB points. All stimuli were gated on and off with quarter-sine and quarter-cosine ramps, respectively, with a duration of 2.5 ms (between 0 and 1). The offset of the masker and the onset of the signal were cross-faded at the -3 -dB points. The signal was presented at 10 dB above the signal detection threshold in quiet for each participant when the elicitor was not present and at 10 dB above the signal detection threshold in the presence of the elicitor for each of the elicitors. The masker level was varied adaptively. The ipsi-long and the contra-long elicitors both had a duration of 500 ms and the ipsi-short elicitor had a duration of 15 ms. The offset of the elicitor and the onset of the masker were cross-faded at the -3 -dB points. The ipsi-long and contra-long elicitors were both presented at 36.2 dB SPL/ERB (40 dB SPL overall level) to match the elicitor level used by Yasin et al. (2014). The level of the ipsi-short elicitor was set individually so that it produced the same amount of masking of the signal as the ipsi-long elicitor. To determine this, the signal level was set to its detection threshold in the presence of the ipsi-long elicitor and the level of the ipsi-short elicitor was adjusted until it just masked this signal.

All stimuli were generated digitally at a sampling rate of 24.414 kHz using TDT System 3 (Tucker-Davies Technologies, Alachua, FL, USA) and MATLAB (The Mathworks, Natick, MA, USA). They were digital-to-analogue converted with a 24-bit amplitude resolution (TDT RP2), amplified (TDT HB7), and presented through Sennheiser HD 600 headphones (Wedemark-Wennebostel, Germany) in a double-walled, sound-attenuating booth (IAC, Winchester, UK).

4.2.2.4 Statistical analysis

The data were analysed using LMMs, implemented in R (R Core Team, 2012). The models were constructed using the forward selection method. The model parameters were fitted using the *lme* function, which is part of the *nlme* package for R (Pinheiro J, 2014). Spearman correlations were calculated between the cochlear gain without the elicitor (G_{max}) and elicitor-induced change in cochlear gain (ΔG). The correlations were calculated using the *rcorr* function, which is part of the *Hmisc* package for R (Harrell et al., 2014).

4.2.3 RESULTS

Participant	Signal quiet threshold (dB SPL)	Signal level ipsi-long elicitor (dB SPL)	Signal level contra-long elicitor (dB SPL)	Level of ipsi-short elicitor (dB SPL)
L1	30 (± 0.4)	46 (± 1.3)	32 (± 0.5)	68 (± 2.3)
L2	25 (± 0.3)	42 (± 0.8)	27 (± 0.7)	77 (± 0.9)
L3	28 (± 1.5)	40 (± 0.4)	29 (± 2.2)	66 (± 2.4)
L4	29 (± 0.5)	42 (± 0.5)	28 (± 0.6)	75 (± 1.3)
L5	29 (± 0.3)	43 (± 1.2)	30 (± 1)	86 (± 1.8)
Mean	28.2 (± 0.9)	42.6 (± 1.0)	29.2 (± 0.9)	74.4 (± 3.6)

Table 4.1: *The signal detection threshold in quiet, in the presence of the ipsi-long elicitor, and in the presence of the contra-long elicitor is listed as well as the level needed for the ipsi-short elicitor to produce the same amount of masking as the ipsi-long elicitor. The SEM is shown in brackets beside each threshold.*

The lowest level at which the elicitor evoked the MEMR was, on average, 83 dB SPL (± 3.0 dB) for the ipsilateral noise and 82 dB SPL (± 4.6 dB) for the contralateral noise. The lowest elicitor level at which the MEMR was elicited in any participant for the ipsilateral elicitor was 75 dB SPL, and, for the contralateral elicitor, was 70 dB SPL. This means that it is unlikely that the ipsi-long or contra-long elicitors, which were presented at 40 dB SPL, elicited the MEMR (see the Chapter 3, Section 3.2.3). The ipsi-short

elicitor, however, was presented at 74.4 (\pm 3.6) dB SPL on average across all participants and the highest ipsi-short elicitor level used was 89.8 dB SPL (see Table 4.1). It is thus possible that the ipsi-short elicitor was loud enough to elicit the MEMR in some participants. However, the ipsi-short elicitor was probably too short to elicit the MEMR in time to affect the masker (Church and Cudahy, 1984) and the MEMR has little or no effect on sounds above \sim 2 kHz (Nuttall, 1974), so is also unlikely to have affected the signal.

4.2.3.1 Without-elicitor TMCs

Figure 4.2A shows the on- and off-frequency FDMCs without the elicitor, averaged across all participants. No significant difference was found between the off-frequency FDMCs for the with- and without-elicitor conditions [tested with LMM analysis, with elicitor condition (present, absent) as a fixed factor, signal duration as a covariate, and participants as random intercepts; main effect and all interactions: ipsi long: $\chi^2(1) = 1.941, p = 0.164$; ipsi short: $\chi^2(1) = <0.000, p = 0.999$; contra long = $\chi^2(1) = 0.988, p = 0.320$]. The off-frequency masker FDMC was therefore averaged across all with- and without-elicitor conditions (shown in Figure 4.2A). The off-frequency masker level at threshold grew approximately linearly with increasing signal duration, as expected. The growth rate of the off-frequency masker level at threshold should correspond to the rate of decrease of the masker effectiveness with decreasing masker duration. Somewhat unexpectedly, the on-frequency masker level at threshold grew at a slightly shallower rate than for the

off-frequency masker level at threshold for signal durations between 2.5 ms and 7.5 ms, although the growth rate became steeper between 7.5 ms and 12.5 ms.

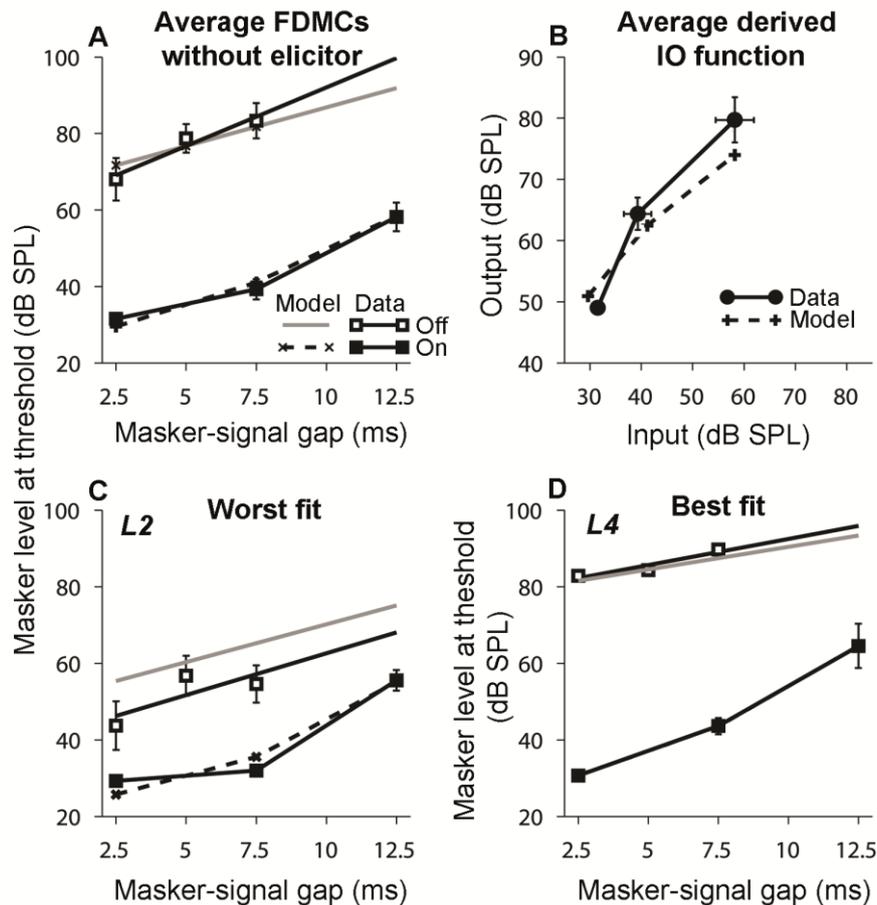


Figure 4.2: Panel A shows the measured (solid lines) and fitted (dotted and grey lines) on- and off-frequency FDMCs (filled and open squares and plus and cross symbols, respectively) without the elicitor present, averaged across all participants. The off-frequency FDMCs are the average of the with- and without-elicitor conditions (see Section 4.2.3.1). Panel B shows the IO functions derived from these data. Panels C and D show the measured and fitted FDMC data for the individuals for whom the model

produced the worst and best fit. The error bars show the standard error of the mean (SEM). The full dataset is shown in Appendix B (Figure 1).

In previous studies, the parameters of the on-frequency masker IO function (maximum gain, G_{max} , and compression exponent, c) were read out directly from the FDMCs (see Yasin et al., 2014). Here, the same approach was taken as in Chapter 3 in that the parameters were fitted using a generic model of the signal and masker responses. As in Chapter 3, the signal and on-frequency masker responses (“excitation”) were assumed to be equal to a piecewise linear function, f_a , of the relevant pressure level, L , and the off-frequency masker excitation was assumed to be equal to a linear function, f_p , of the masker pressure level (see Chapter 3, Section 3.1.3.2). The masking effectiveness, ME , was assumed to decay exponentially, at a rate τ , with increasing signal duration (and decreasing masker duration), S_{dur} : $ME(S_{dur}) = ME(S_{dur} = 0) \cdot e^{-S_{dur}/\tau}$. Further, it was assumed that the masker level at threshold corresponded to a constant ratio, k , between the signal and masker excitation. Using these assumptions, the on- and off-frequency masker levels at threshold, L_{on} and L_{off} , for each signal duration, S_{dur} , were predicted as:

$$L_{on}(S_{dur}) = f_a^{-1} \left(10^{L_{ss}/10} \cdot 10^{k/10} / e^{-S_{dur}/\tau} \right)$$

and $L_{off}(S_{dur}) = f_p^{-1} \left(10^{L_{ss}/10} \cdot 10^{k/10} / e^{-S_{dur}/\tau} \right)$, where f_a^{-1} and f_p^{-1} are the inverse of the functions f_a and f_p , and L_{ss} is the signal sensation

level. As in Chapter 3, the sum of squared differences between all predicted and observed masker levels at threshold (on- and off-frequency) was minimised by varying the maximum gain, G_{max} , the compression exponent, c , the first break point, BP_1 , the signal-to-masker ratio, k , and the masker decay rate, τ . The model was fitted to each participant's data separately. Unlike in Chapter 3, the passive off-frequency masker attenuation, P , was fixed at 20 dB. This value matched the average passive attenuation measured in the previous study and the average attenuation in Yasin et al. (2014)'s TMC and FDMC data (estimated by subtracting the on- from the off-frequency masker levels at threshold at large masker-signal gaps/ signal durations, where the on- and off-frequency masking curve growth rates had converged).

The model produced a good fit to the data (Figure 4.2). The RMSD between the average predicted and observed masker levels at threshold was only 2.12 dB (Figure 4.2A, B). The RMSD between the individual predicted and observed masker levels at threshold ranged from 0.60-4.35 dB; panels C and D in Figure 4.2 show the worst- and best-fit datasets. An LMM analysis, with masking condition (on- and off-frequency) and data type (predicted, observed) as fixed factors, signal duration as covariate, and participants as random intercepts, showed that the predicted thresholds were not significantly different from the observed thresholds [main effect and all interactions of data type: $\chi^2(2) = 0.162$, $p = 0.92$]. On average, the maximum gain, G_{max} , was estimated as 22.1 ± 3.57 dB, the compression exponent, c , as 0.37 ± 0.091 , and the

lower breakpoint, BP_1 , as 43.8 ± 3.07 dB. Across individuals, G_{max} ranged from 9.7 to 30.8 dB, and c ranged from 0.23 to 0.73 (see Figure 2.4A, B; all parameters are reported in Appendix B, Table 1).

4.2.3.2 Effect of the elicitor

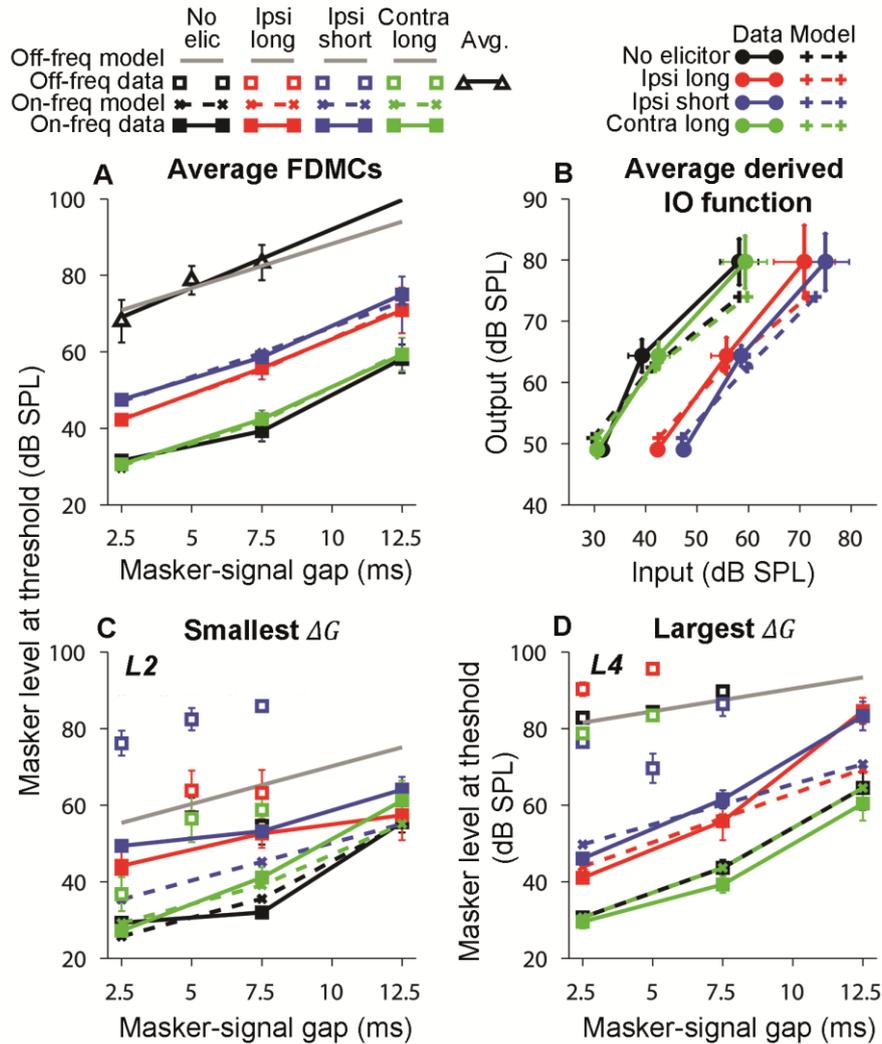


Figure 4.3: Panel A shows the FDMCs, averaged across all participants. Measured (solid lines) and fitted (dotted and grey lines) FDMCs are shown for the on- and off-frequency conditions without the elicitor and for the on-frequency condition only with the elicitor (see legend). As in Figure 4.2, for

the off-frequency condition, the average FDMC data across all elicitor conditions is shown (see Section 4.2.3.1). Panel B shows the IO functions derived from these FDMCs. Panels C and D show the measured and fitted FDMCs for the participants who had the largest and smallest maximum change in ΔG caused by any of the elicitors. In these panels the off-frequency data without the elicitor and with each elicitor is shown (see legend). The error bars show the SEM. The full dataset is shown in Appendix B (Figure 1).

Panels A and B in Figure 4.3 show the average data from all participants. The ipsi-long and ipsi-short elicitors increased the on-frequency masker levels at threshold substantially across all signal durations. The contralateral elicitor, however, had little or no measurable effect. The quiet detection threshold of the 2.5-ms, 4-kHz sinusoidal signal was 28.2 ± 0.9 dB SPL, on average (see Table 4.1). This increased to 42.6 ± 1.0 dB SPL, on average, when the ipsi-short or ipsi-long elicitor was present and to 29.2 ± 0.9 dB SPL, on average, when the contra-long elicitor was present. The signal detection threshold in quiet and with the contra-long elicitor were no significantly different [paired t -test: $t(4) = 1.8, p = 0.141$].

An LMM analysis of the data from all participants showed that the effect of the ipsi-long elicitor had a significant main effect on the on-frequency masker levels at threshold [$\chi^2(1) = 22.438, p = < 0.001$]. The interaction between elicitor condition and signal duration was not significant [$\chi^2(1) = 0.123, p = 0.726$]. Similarly, the ipsi-short elicitor also had a significant

main effect on the on-frequency masker levels at threshold [$\chi^2(1) = 35.241$, $p = < 0.001$], but, again, the effect did not interact significantly with signal duration [$\chi^2(1) = 0.0299$, $p = 0.863$]. Finally, unlike for the ipsi-long or ipsi-short elicitors, the contra-long elicitor did not have any significant effect on the on-frequency masker levels at threshold [main effect and all interactions: $\chi^2(1) = 0.583$, $p = 0.445$].

The with-elicitor thresholds were fitted with the same model as was used for the without-elicitor thresholds. The first and second break points, BP_1 and BP_2 , the signal-to-masker ratio, k , and the masker decay rate, τ , were carried over from the without-elicitor fits. As in Chapter 3, only G_{max} was allowed to vary. G_{max} was fitted by minimizing the sum of squared differences between all predicted and observed with-elicitor thresholds. The fitting was conducted separately for each participant and elicitor.

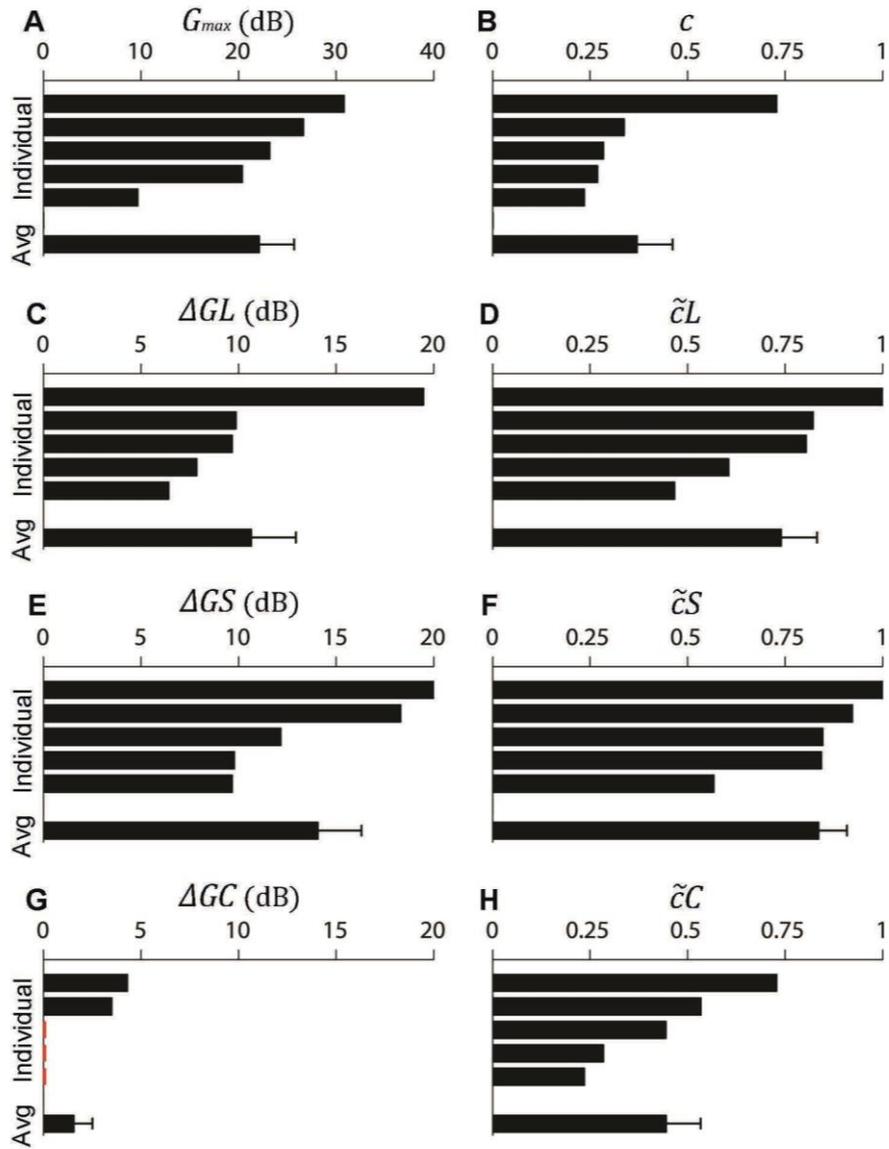


Figure 4.4: Individual model parameter estimates for the maximum cochlear gain (G_{max}) and the compression exponent without the elicitor (c) are shown (panels A and B, respectively). The reduction in gain (ΔG) and compression exponent (c) caused by the ipsi-long, (ΔGL and $\tilde{c}L$, panels C and D), ipsi-short (ΔGS and $\tilde{c}S$, panels E and F) and contra-long elicitors (ΔGC and $\tilde{c}C$, panels G and H) is also shown. Both individual and average

parameter values are shown. Parameter values of zero are marked by a red line.

Despite the fact that only one parameter was fitted, the model produced a good fit to the thresholds in each of the with-elicitor conditions (see Figure 4.3). For the ipsi-long elicitor, the RMSD between the average predicted and observed with-elicitor thresholds was 2.50 dB, and ranged from 1.97 to 6.38 dB across individuals. For the ipsi-short elicitor, the average RMSD was 3.28 dB, and ranged from 4.13 to 8.06 dB, and for the contra-long elicitor, the RMSD was 1.84 dB on average, and ranged from 2.50 to 4.43 dB. LMM analyses, with masking condition (on, off) and data type (predicted, observed) as fixed factors, signal duration as covariate, and participants as random intercepts, showed that the predicted and observed thresholds were not significantly different for any of the elicitors [ipsi long: $\chi^2(2) = 2.992, p = 0.224$; ipsi short: $\chi^2(2) = 2.813, p = 0.245$; contra long: $\chi^2(2) = 0.296, p = 0.862$]. For the ipsi-long and ipsi-short elicitor, allowing to G_{max} to vary yielded a significantly better fit to the with-elicitor data than fixing G_{max} at the value fitted to the without-elicitor thresholds [tested with an F -test; ipsi long: $F(1,2) = 57.81, p = 0.017$; ipsi short: $F(1,2) = 38.38, p = 0.025$]. On average, the ipsi-long elicitor caused G_{max} to decrease by 10.65 ± 2.293 dB, the ipsi-short elicitor caused G_{max} to decrease by 14.07 ± 2.241 dB, and the contra-long elicitor caused G_{max} to decrease by 1.56 ± 0.963 dB. A paired t -test showed that the elicitor-induced change in G_{max} (ΔG , see Figure 4.1D) was not significantly different between the ipsi-long and ipsi-short

elicitors, although this was marginal [$t(4) = 2.3, p = 0.080$]. Across individuals, the elicitor effect on G_{max} , showed a considerable degree of variability. For the ipsi-long elicitor, the elicitor-induced change in G_{max} (ΔG) ranged from 6.41 to 19.47 dB, for the ipsi-short elicitor, ΔG ranged from 9.66 to 20.48 dB, and for the contra-long elicitor, the ΔG ranged from 0 to 4.30 dB.

As in Chapter 3, there was no significant correlation between G_{max} without the elicitor and the elicitor-induced change in G_{max} , ΔG for any of the elicitors (ipsi long: $r = -0.10, p = 0.873$; ipsi short: $r = 0.50, p = 0.391$; contra long: $r = -0.11, p = 0.858$). There was also no correlation between G_{max} and the signal detection threshold in quiet ($r = -0.89, p = 0.162$), or between ΔG and the elicitor effect on the signal detection threshold in quiet for any of the elicitors (ipsi long: $r = 0.30, p = 0.624$; ipsi short: $r = 0.00, p = 1.000$; contra long: $r = 0.86, p = 0.061$). Correlations have not been corrected for multiple comparisons between elicitors so that the results are comparable to previous studies which often only had one elicitor.

4.3 EXPERIMENT 2: SUPPRESSION OF OTOACOUSTIC EMISSIONS (OAEs)

4.3.1 GENERAL OUTLINE AND EXPECTATIONS

In this experiment, suppression of OAEs evoked by a probe stimulus was measured for a long and short elicitor presented ipsilateral to the probe (ipsi long and ipsi short) and for a long elicitor presented contralateral to the probe (contra long). The elicitors had the same temporal characteristics as the elicitors used in Experiment 1. A contralateral elicitor that was presented simultaneously with the probe was also used so that OAE suppression by the ipsi-long, ipsi-short and contra-long elicitors could be compared to a more commonly used elicitor of OAE suppression. The probe was a click and was presented at a level of either 60 or 70 dB pe SPL. Using two different probe levels allows IO suppression to be measured as well as amplitude suppression (see Chapter 3, Section 3.3.1). Experiment 2 was conducted after Experiment 1, with the same group of participants. As discussed in Chapter 3, neither amplitude nor IO suppression of OAEs would be expected to represent a quantitative measure of MOCR-induced reduction in cochlear gain. However, either or both OAE suppression measures might be expected to correlate with cochlear-gain change estimated in Experiment 1 (ΔG).

4.3.2 METHODS

4.3.2.1 OAE measurements

Click-evoked OAEs were recorded using a system that was custom built for this experiment. All stimuli were generated digitally at a sampling rate of 24.414 kHz using TDT system 3 (Tucker-Davies Technology, Alachua, FL, USA) and MATLAB (The Mathworks, Natick, MA, USA). They were digital-to-analogue converted with a 24-bit resolution (TDT RP2) and amplified (TDT HB7). The clicks had a 100- μ s duration and were presented to the participant's right ear. They were presented at a rate of 20/s using a general-purpose OAE transducer (Otodynamics, Hatfield, UK).

OAEs were measured with and without either the ipsi-short, ipsi-long, contra-long, or contra-sim elicitor present. The duration of the ipsi-short, ipsi-long, and contra-long elicitors was the same as in Experiment 1 and the contra-sim elicitor had the same duration as the contra-long elicitor. The ipsi-short and ipsi-long elicitors were presented ipsilaterally to the click, and the contra-long and contra-sim elicitors were presented contralaterally. The centre frequency of the elicitors was set individually at the frequency where the amplitude of the click-evoked OAE without the elicitor was largest; the elicitor centre frequencies were: 1.5 kHz (L1), 1.5 kHz (L2), 1.4 kHz (L3), 1.7 kHz (L4) and 1 kHz (L5). The elicitors were all 20-ERBs-wide noises and were presented at the same level per ERB as in Experiment 1.

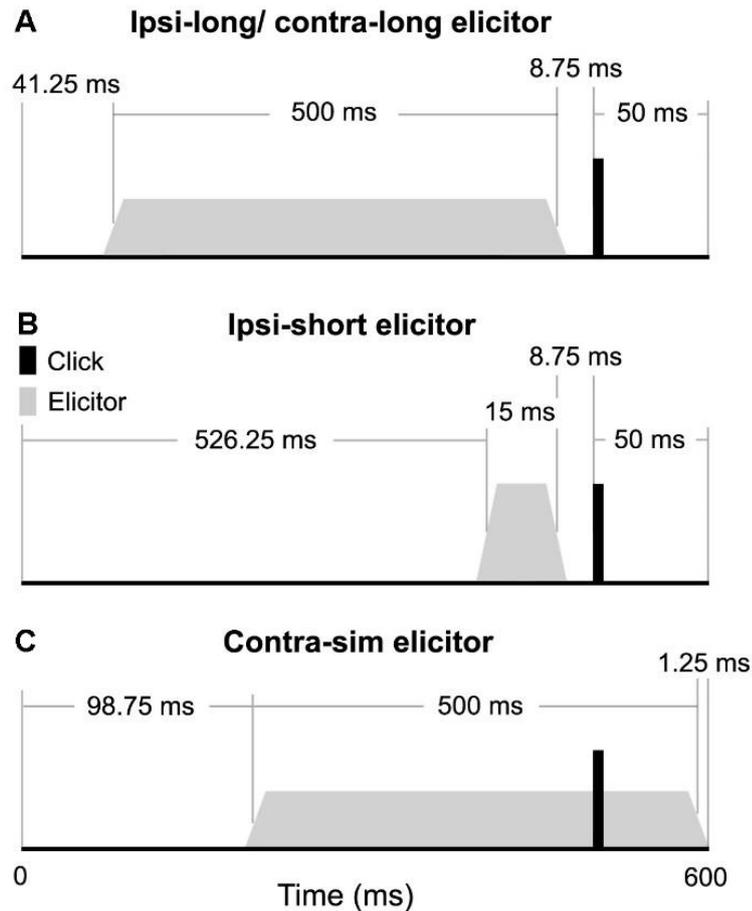


Figure 4.5: Panels A, B and C show the temporal characteristics for the ipsi-long and contra-long, the ipsi-short, and the contra-sim elicitor conditions used in Experiment 2, respectively. The probe click is shown in black and the elicitor in grey. Stimuli are not plotted to scale.

Each trial was 600-ms long. The temporal relationship between each of the elicitors and the click is shown in Figure 4.5. The click was always presented 50 ms from the end of the trial to allow time for the OAE to be recorded. The gap between the elicitor offset (3-dB down) and click onset was 8.75 ms for all elicitors apart from the contra-sim elicitor, the offset of which coincided with the end of the trial. The 8.75-ms gap between the

offset of the ipsi-long, ipsi-short and contra-long elicitors and the click was chosen so that the click would occur at the same time as the temporal centre of the longest masker used in Experiment 1. The temporal relationship between the elicitor and the click was the same for the ipsi-long and contra-long elicitors.

The OAEs were recorded using the transducer microphone and were amplified by a factor of 20,000, low-pass filtered at 10,000 Hz (24 dB/octave roll-off) and high-pass filtered at 300 Hz (12 dB/octave roll-off) using an ICP511 alternating-current difference amplifier (Grass Telefactor, West Warwick, RI). The OAEs were analogue-to-digital converted and averaged using the TDT RP2. They were averaged online over 2000 trials. Two such averages (“replicates”) were recorded for each click level (60 and 70 dB pe SPL) and elicitor condition. Trials were rejected if the response amplitude exceeded 5 mPa within the period from 6-16 ms after the click.

The ipsi-long and ipsi-short elicitors were measured in one session and the contra-long and contra-short elicitors in another session. These sessions were completed on a separate days. Participants L2 and L5 completed the session with the ipsi-long and ipsi-short elicitors first, and the remaining three participants completed the sessions in the reverse order. Each session contained 14 blocks, which each lasted ~7 mins and were separated by ~30 s of silence. Participants were given a break of at least 10 minutes after seven blocks. In each block, OAEs were recorded

with and without the elicitors in a random order. A new, randomly generated frozen-noise was used for the elicitor in each block. Within a single block, 200 trials were alternately recorded into one of two buffers so that, in total, two 100-trial “replicates” were obtained. This was repeated seven times in each session so that 700 trials were completed for each replicate. The OAE measurements were performed in the same sound-attenuating booth as Experiment 1. Participants watched a silent subtitled movie of their own choice to stay alert.

4.3.2.2 OAE data analysis

Offline-analysis of OAEs was performed in MATLAB. First, the OAEs were further band-pass filtered from 750-4000 Hz by applying a 2nd-order Butterworth bandpass filter in both the forward and reverse time direction to create zero phase delay. As in Chapter 3, the OAEs were windowed between 6-16 ms after the click and the window edges were rounded according to 2-ms quarter-sine and -cosine functions. The OAE amplitude for each condition was taken as the integral of the co-spectrum between the respective replicates. The co-spectrum is the real part of the spectrum. OAEs were accepted as valid only if the correlation between the two replicates for each condition, referred to as “reproducibility”, was >0.7. The reproducibility for all included OAEs averaged 0.91 (± 0.009). The methods used for the statistical analysis of the OAE data were the same as those used in Experiment 1.

4.3.3 RESULTS

As discussed Section 4.2.3, the lowest level at which the elicitor evoked the MEMR in any participant was 75 dB SPL for the ipsilateral noise and 70 dB SPL for the contralateral broadband (0.125– 4 kHz) noise. It is unlikely that the ipsi-long, contra-long, or contra-sim elicitors used in this experiment, which were 50 dB SPL (louder than in Experiment 1 because of the larger elicitor bandwidth used, although the same level in dB per ERB as in Experiment 1), elicited the MEMR (see Chapter 3, Section 3.2.3). The ipsi-short elicitor used in this experiment, however, was presented at 88.2 dB SPL on average, and the highest ipsi-short elicitor level used was 99.8 dB SPL (L5). As in Experiment 1, this elicitor may have been loud enough to elicit the MEMR, but was probably too short to elicit the MEMR in time to affect the probe.

4.3.3.1 OAE suppression

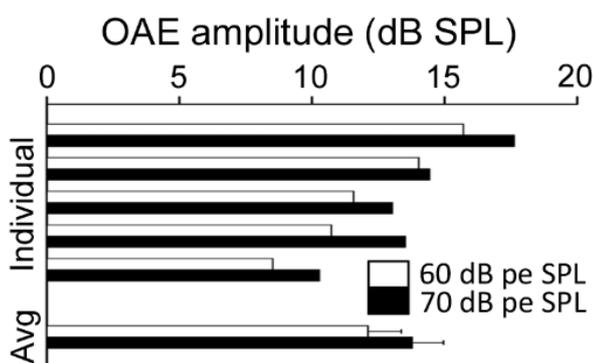


Figure 4.6: Individual and average OAE amplitudes without the elicitor are shown for the 60- and 70-dB pe SPL clicks (white and black bars respectively). For the average data, error bars show the SEM.

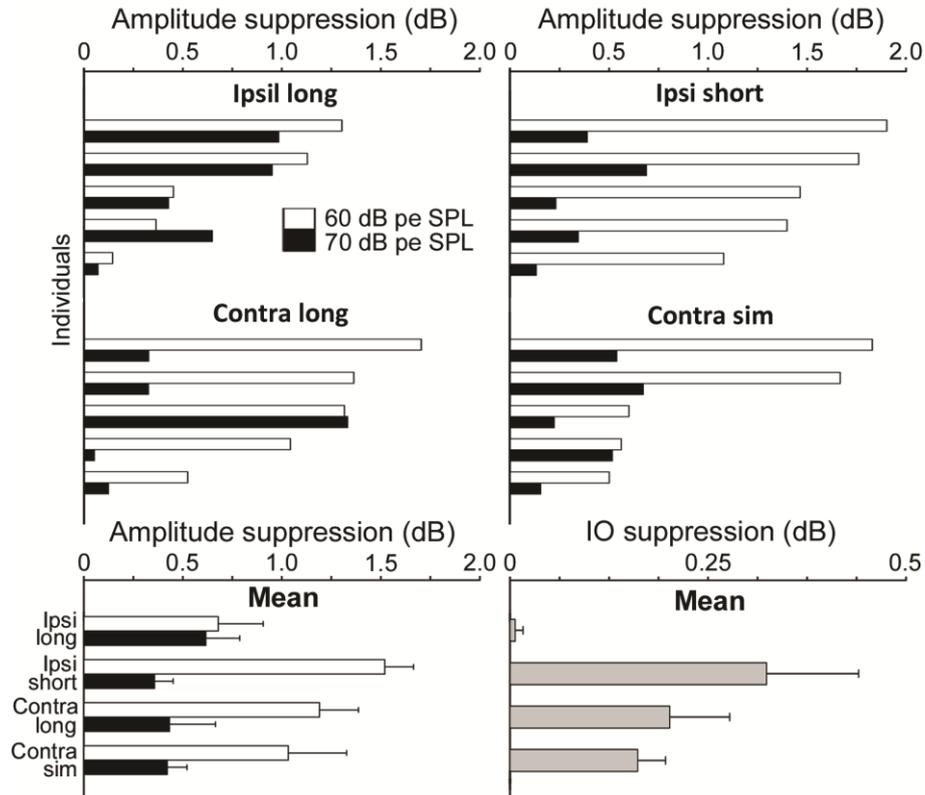


Figure 4.7: In the top panel, individual and average OAE amplitude suppression by the ipsi-long, ipsi-short, contra-long and contra-sim elicitors is shown for the 60- and 70-dB pe SPL clicks (white and black bars respectively). In the lower panel, average amplitude suppression (left) and IO suppression (right) for each elicitor is shown. For the average data, error bars show the SEM.

The OAE amplitude was larger for the 70 dB pe SPL clicks than for the 60 dB pe SPL clicks; this effect of click level and the overall OAE amplitude was not different between the ipsilateral and contralateral elicitor sessions [Figure 4.6; tested with LMM analysis, with session (ipsilateral, contralateral) and click level as fixed factors and participants as random intercepts; main effect of click level: $\chi^2(1) = 8.704, p = 0.003$; main effect

of session: $\chi^2(1) = 0.771, p = 0.380$; interaction between click level and session: $\chi^2(1) = 0.261, p = 0.610$]. Furthermore, the OAE amplitude was smaller with than without the elicitor for all of the elicitors [tested with separate LMM analyses, with elicitor (present, absent) and click level as fixed factors and participants as random intercepts; main effect of elicitor, ipsi long: $\chi^2(1) = 8.545, p = 0.003$; ipsi short: $\chi^2(1) = 10.244, p = 0.001$; contra long: $\chi^2(1) = 6.594, p = 0.010$; contra sim: $\chi^2(1) = 5.127, p = 0.024$]. IO suppression, whereby the elicitor effect is larger for the 60- than 70 dB pe SPL click, was found for the ipsi-short, contra-long and contra-sim elicitors [see Figure 4.7; ipsi short: $\chi^2(1) = 22.047, p = < 0.001$; contra long: $\chi^2(1) = 6.314, p = 0.012$; contra sim: $\chi^2(1) = 5.103, p = 0.024$] but not for the ipsi-long elicitor [$\chi^2(1) = 0.464, p = 0.496$]. For the 60 dB pe SPL click, for which the largest amplitude suppression was found for all but one elicitor, the elicitor caused an average reduction in OAE amplitude of 0.68 dB (ranging from 0.15 to 1.30) for the ipsi-long elicitor, 1.52 dB (ranging from 1.08 to 1.90) for the ipsi-short elicitor, 1.19 dB (ranging from 0.52 to 1.70) for the contra-long elicitor, and of 1.03 dB (ranging from 0.50 to 1.83) for the contra-sim elicitor. The average IO suppression was 0.006 dB (ranging from -0.028 to 0.032) for the ipsi-long elicitor, 0.116 dB (ranging from 0.095 to 0.151) for the ipsi-short elicitor, 0.076 dB (ranging from -0.002 to 0.138) for the contra-long elicitor, and 0.035 dB (ranging from 0.005 to 0.129) for the contra-sim elicitor. The ipsi-short elicitor caused significantly more IO suppression than the ipsi-long elicitor [$t(4)=6.8, p = 0.002$], but the amount of

amplitude suppression for the 60 dB pe SPL click was not significantly different [$t(4) = 2.8, p = 0.103$; corrected for multiple comparisons].

4.3.3.2 Correlation with psychophysical data

Two-tailed Spearman correlations were calculated between the elicitor-induced reduction in cochlear gain, ΔG , measured psychophysically in Experiment 1 and the OAE measurements of the elicitor effect measured in Experiment 2 (Figure 4.8). It was found that the OAE amplitude suppression did not correlate significantly with ΔG for either the ipsi-long ($r = 0.10, p = 0.873$; Figure 4.8A), ipsi-short ($r = 0.00, p = 1.000$; Figure 4.8B), or contra-long ($r = -0.34, p = 0.581$; Figure 4.8B) elicitors. The IO suppression showed a significant correlation with ΔG only for the contra-long elicitor (see Figure 4.8, left panels; contra long: $r = 0.89, p = 0.040$; ipsi long: $r = 0.20, p = 0.7471$; ipsi short: $r = -0.50, p = 0.391$).

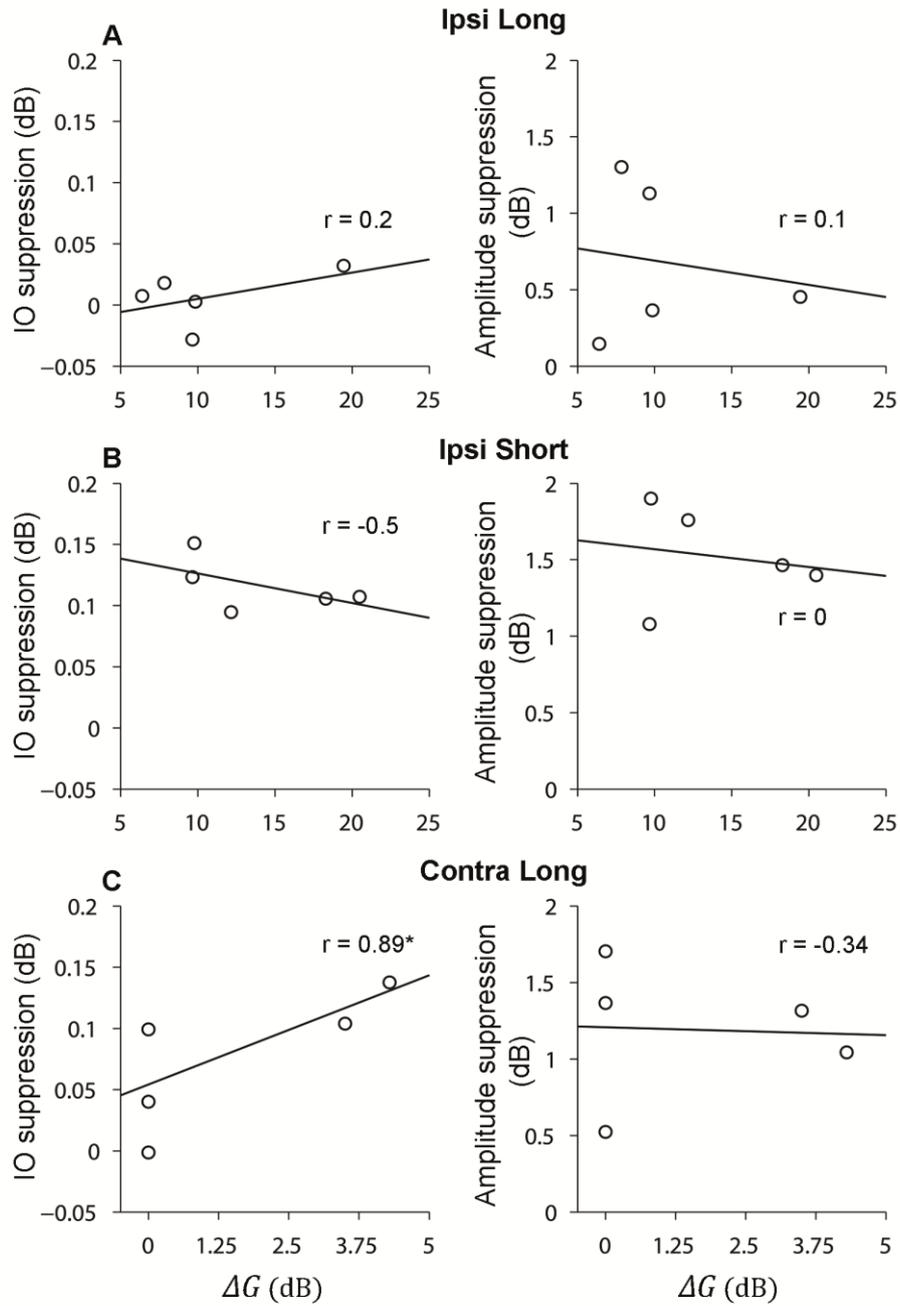


Figure 4.8: Correlations between OAE estimates of IO suppression (left) and amplitude suppression (right) for each elicitor and psychophysical estimates of cochlear-gain reduction (ΔG). Correlations are shown for ipsi-long (panel A), ipsi-short (panel B), and contra-long (panel C) elicitors. In each panel, the Spearman's rank correlation coefficient (r) and a linear regression fit (solid line) are shown.

4.4 DISCUSSION

In a recent study, Yasin et al. (2014) found a large psychophysical masking effect caused by an ipsilateral narrowband-noise elicitor, which they attributed to an MOCR-induced cochlear-gain reduction. To test whether this effect was due to the MOCR, the amount of cochlear gain reduction caused by three different elicitors was estimated using a very similar FDMC method as used by Yasin et al. (2014). In the current study, cochlear gain, cochlear compression, and cochlear-gain reduction by the elicitor were estimated from the FDMCs by fitting the entire dataset with a generic model of the cochlear IO function. Furthermore, OAE suppression by the elicitors was measured to independently verify whether the elicitors had caused a reduction in cochlear gain. Firstly, the large effect that Yasin et al. (2014) found for a long-duration narrowband-noise elicitor presented ipsilateral to the masker and signal was replicated. This ipsi-long elicitor also produced a statistically significant OAE amplitude suppression, but no significant IO suppression. The second elicitor used here (contra-long) was the same as the ipsi-long elicitor, but was presented contralateral to the signal and masker. Because the MOCR can be elicited by contralateral sounds, this elicitor was also expected to produce a measurable cochlear-gain reduction. However, while the elicitor caused a statistically significant OAE amplitude and IO suppression, indicating that a cochlear-gain reduction did occur, no effect on the psychophysical estimates of cochlear gain was found. OAE suppression effects were similar whether

the elicitor was presented slightly before the probe (contra-long), or, as in most previous studies using OAEs, was presented simultaneously with the probe (contra-sim). Finally, the third elicitor used (ipsi short) was set to cause the same amount of masking as the ipsi-long elicitor, but was thought to be too short to elicit the MOCR in time to affect the masker effectiveness. Therefore, if the effect of the ipsi-long elicitor was due to MOCR-induced cochlear-gain reduction, the ipsi-short elicitor would have been expected to produce no effect. However, the psychophysical estimate of the cochlear-gain reduction for the ipsi-short elicitor was similar or larger than for the ipsi-long elicitor. Furthermore, the ipsi-short elicitor produced a significant OAE amplitude and IO suppression.

The absence of an effect of the contra-long elicitor in Experiment 1 was surprising, considering that the OAE estimates suggest that the effect of the contra-long elicitor on cochlear gain was similar to the effect of the ipsi-long elicitor, which produced a large effect in Experiment 1. However, the OAE estimates from Experiment 2 were made using noise elicitors that were more broadband than in Experiment 1 and there is evidence that the relative strength of the ipsilateral and contralateral reflex depends on the bandwidth of the elicitor (see Chapter 1, Section 1.1.3). Using a noise elicitor with a similar bandwidth to the one used in Experiment 1, Lilaonitkul and Guinan (2009a) found that the amount of cochlear-gain reduction may be up to twice as large when the elicitor is presented ipsilaterally, rather than contralaterally. However, even if the contralateral reflex was half as strong as the ipsilateral reflex, assuming

the effect produced by the ipsi-long elicitor was due to MOCR-induced cochlear-gain reduction, a cochlear-gain reduction by the contra-long elicitor of ~5 dB would still be expected.

The absence of a measurable effect by the contra-long elicitor used in the current study accords with the finding of a 4-dB cochlear-gain reduction by a contralateral elicitor found in Chapter 3. The elicitor used in Chapter 3 was > 3.5 octaves wide, whereas the one used in the current study and by Yasin et al. (2014) was < 0.5 octaves wide. According to the findings of Lilaonitkul and Guinan (2009a), the broader bandwidth of elicitor used in Chapter 3 would be expected to cause more than twice as much MOCR-induced cochlear-gain reduction than the elicitor used in the current study. Furthermore, the elicitor used in the current study was more than 10 dB quieter than the one used in Chapter 3 and would thus be expected to be a weaker MOCR elicitor. The narrower bandwidth and lower level of the elicitor used in the current study can therefore account for the absence of a measurable effect. The idea that the contralateral elicitor produced no reduction in cochlear gain is also supported by the absence of an effect of the elicitor on the signal detection threshold (although see Section 2.4 for a discussion of how a change in cochlear gain may not always affect signal detection thresholds). It remains puzzling, however, as to why the contra-long elicitor caused significant OAE amplitude suppression.

The ipsi-short elicitor produced a large reduction in both psychophysical (Experiment 1) and OAE (Experiment 2) estimates of cochlear gain and this effect was of a similar magnitude to the effects measured for the ipsi-long elicitor. In fact, there is some suggestion in both the OAE and psychophysical data that the effect of the ipsi-short elicitor may be slightly larger than the ipsi-long elicitor. This may be a result of the large within-participant variability in the ipsi-short elicitor level required to create the same amount of masking as the ipsi-long elicitor.

The gap between the onset of the ipsi-short elicitor and the probe click used to evoke the OAEs in Experiment 2 was 23.75 ms and the shortest gap between the onset of the elicitor and the temporal centre of the masker in Experiment 1, was 18.75 ms. The fastest MOCR effects that have been found using OAEs occurred between 25-95 ms after the elicitor onset and the effects did not reach their maximum until at least ~80 ms after the elicitor onset (James et al., 2002a; Backus and Guinan, 2006). However, as discussed in Chapter 1, physiological studies in animals have found some MOCR fibres that responded within ~5 ms of the onset of an elicitor. It therefore remains possible that the effect of the ipsi-short elicitor was due, at least partially, to an MOCR-induced reduction in cochlear gain and that the MOCR is faster than has previously been assumed.

As discussed in the Methods sections of Experiments 1 and 2, the ipsi-short elicitor was presented at a high level and it was thus not possible

to conclusively rule out elicitation of the MEMR. However, it was argued that the elicitor was likely too short to elicit the MEMR in time to effect the off-frequency masker in Experiment 1 and that the signal and on-frequency masker were too high in frequency to be affected. The finding of strong IO suppression by the ipsi-short elicitor in Experiment 2 suggests that this effect was also not due to the MEMR, which would be expected to affect both probe levels approximately equally (Pang and Guinan, 1997).

As discussed in Section 4.1, the effect of both the ipsi-short and ipsi-long elicitors may have been due a phenomenon referred to as temporal suppression, whereby the OAE amplitude for a probe click is reduced by a preceding “suppressor” click. Temporal suppression depends on the level of the suppressor and probe, and is thought to be caused by a reduction in the amount of cochlear gain applied to the probe. This cochlear-gain reduction is thought to arise as a result of the persistence of the basilar membrane response to the suppressor click, which disrupts the mechanisms of cochlear amplification for the probe and thereby reduces the cochlear response to the probe (Kapadia and Lutman, 2000a, b; Harte et al., 2005). One feature of temporal suppression that links it to persistence of basilar membrane motion is that it decays fairly rapidly (within ~10 ms of the offset of the suppressor click; e.g., Kapadia and Lutman, 2000a; Verhulst et al., 2011; Bianchi et al., 2013). Moreover, the decay of temporal suppression with increasing gap between the suppressor click and the probe has been shown to be frequency

dependent (Bianchi et al., 2013): at 4 kHz, temporal suppression decays between ~2-4 ms after the suppressor click, whereas at 2 kHz, it decays between ~4-8 ms after the suppressor click. This would be expected, as the basilar membrane response persists longer at low than at high frequencies (e.g., Recio and Rhode, 2000). In Experiment 2, the offset of the ipsi-short and ipsi-long elicitors occurred 8.75 ms before the onset of the probe and, in Experiment 1, the maximum gap between the offset of the elicitor and the temporal centre of the masker was also 8.75 ms. In Experiment 1, cochlear-gain reduction estimates were made at 4 kHz. Bianchi et al. (2013) found that a 65 dB pe SPL click caused little temporal suppression at 4-kHz 8-ms after its offset. However, the ipsi-short elicitor was presented at 74.4 dB SPL, on average, in Experiment 1, and was much longer than the click used by Bianchi et al. (2013). The ipsi-long elicitor had the same forward masking effectiveness as the ipsi-short elicitor and so both elicitors contained significantly more energy than Bianchi et al. (2013)'s click. Kapadia and Lutman (2000a) found that the amount of temporal suppression increases dramatically with increasing suppressor level, and that temporal suppression persists for much longer at higher suppressor levels. It is therefore possible that at least part of the effects of the ipsi-short and ipsi-long elicitors was due to temporal suppression. In Experiment 1, the elicitor effect was not much decreased at higher masker levels, as may have been expected if the effect was due a reduction in cochlear gain caused by the MOCR (see Figure 4.1). The fact that the effect was similar at low and high masker

levels supports the notion that the gain reduction was caused by temporal suppression. This is because temporal suppression would only be expected to affect the initial part of the masker and would thus be expected to have a greater effect when the masker duration is short (corresponding to high masker levels) than when it is longer (corresponding to low masker levels). Thus, in the case of temporal suppression, the duration effect would be expected to counteract the level effect. Yasin et al. (2014)'s findings suggest that the elicitor may still have some effect when preceding the masker by up to 200 ms, which indicates that the effect of the ipsi-long elicitor is not entirely due to temporal suppression.

As well as the elicitor causing temporal suppression of the masker, the masker may have caused temporal suppression of the signal, particularly at the shortest signal durations. It is not known how temporal suppression depends on the frequency separation between the probe and the suppressor, but if it is due to persistence of basilar membrane motion then it may be expected to occur both with the on- and off-frequency maskers. However, any temporal suppression effect by the masker is likely to have been small because, at short signal durations where the signal would have been most susceptible to temporal suppression, the masker level was low.

It is also possible that the OAE suppression effects by the ipsi-short and ipsi-long elicitors in Experiment 2 were due to temporal suppression. In

Experiment 2, a click probe was used and therefore the OAE amplitude could be affected by cochlear-gain change at a range of frequencies, including lower frequencies where temporal suppression would be expected to persist for longer. Using similar probes to those used in Experiment 2, previous studies suggest that little temporal suppression occurs at any frequency when the suppressor click precedes the probe by 8.75 ms (Kapadia and Lutman, 2000a; Verhulst et al., 2011; Bianchi et al., 2013). However, because the amount of temporal suppression increases with increasing suppressor energy, as for Experiment 1, the high elicitor level and long elicitor duration used means that the ipsi-short and ipsi-long elicitors may have produced at least some temporal suppression of the probe.

As well as the elicitor causing temporal suppression of the masker, the masker may have caused temporal suppression of the signal, particularly at the shortest signal durations. This effect would only be expected in the on-frequency masker conditions and could therefore lead the amount of cochlear gain and compression to be overestimated. However, any temporal suppression effect by the masker is likely to have been minimal because, at the shortest signal durations where temporal suppression might occur, the masker level was always low.

The estimates of maximum cochlear gain and compression when no elicitor was present were highly consistent with previous findings using the FDMC method. According to the model results, the maximum

cochlear gain varied from as little as ~ 10 dB up to ~ 31 dB across the current set of participants. This range is similar to that found in previous studies using both the TMC and FDMC methods (e.g., Yasin et al., 2013b). The average estimated maximum cochlear-gain across all participants in the current study was ~ 22 dB. This is smaller, by ~ 25 dB, than the estimates obtained in previous psychophysical studies (reviewed by Yasin et al., 2013b), but, as discussed in Chapter 3, this is because the current approach of fitting a cochlear IO function model takes account of the passive attenuation of the off-frequency masker at the characteristic place for the signal frequency. The estimated compression exponent without the elicitor was 0.37, on average, and ranged from 0.23 to 0.73. This is similar to previous estimates using the FDMC method (Yasin et al., 2013b, 2014), but greater than the compression exponents typically found with the TMC method (see Chapter 3; Nelson et al., 2001; Yasin et al., 2013b). The current estimate is, however, in line with estimates made in squirrel monkeys and chinchillas, although it is greater than those made in guinea pigs (for review, see Robles and Ruggero, 2001). It seems possible that the compression exponent may have been overestimated in both the current study and in Yasin et al. (2013b, 2014), because, at the shortest signal durations, the masker level at threshold grew more slowly as a function of the signal duration for the on-frequency masker than for the off-frequency masker. This would suggest that the cochlear IO function is expansive at low input levels, which conflicts with previous psychophysical (Oxenham and Plack, 1997; Yasin et al., 2013b) and

physiological (Ruggero et al., 1997) findings. This, coupled with the greater compression exponents found using the FDMC method, suggests that the assumption that the rate of decrease in masking effectiveness with decreasing masker, and increasing signal, duration is the same for on- and off-frequency maskers might not hold in the FDMC method.

In the current study, the model corrected for the unexpectedly shallow growth of the on-frequency compared to off-frequency masker effectiveness by slightly under-predicting the growth of the off-frequency masker levels at threshold with increasing signal duration (see Figure 4.2). In previous studies, however, the compression exponent was calculated directly from the data for the signal durations at which the on-frequency masker level at threshold grows most quickly as a function of signal duration for the on-frequency masker (i.e. at masker levels where compression is maximal). In the current study, a similar estimate can be made by, in both the on- and off-frequency masking conditions, calculating the difference in the average masker level at threshold for a signal duration of 7.5 ms and 12.5 ms separately (Δ_{on} and Δ_{off} , respectively). The compression exponent is then Δ_{off}/Δ_{on} . Using this approach, the estimated compression exponent without the elicitor was 0.59. This is higher than in previous studies using the FDMC method, for which the on-frequency masker levels at threshold and signal and masker durations were similar (Yasin et al., 2013b). One difference between the current study and that by Yasin et al. (2013b), was that, in the current study, a narrowband noise rather than a tonal masker was

used. This was because in pilot measurements, the low on-frequency masker levels at threshold found by Yasin et al. (2013b) could not be replicated with a sinusoidal masker. Low on-frequency masker levels at threshold are desirable because it is at low levels that the largest MOCR effects would be expected. It has previously been shown that a short tonal signal which is at the masker centre frequency, like in the on-frequency masking conditions in the current study, can be confused with fluctuations in the amplitude envelope of a narrowband-noise masker (Moore, 1981; Moore and Glasberg, 1982, 1983; Neff and Jesteadt, 1983). Confusion describes when the signal is detectable, but cannot be discriminated from the masker (Neff, 1986). It is less likely to occur for tonal maskers (Neff and Jesteadt, 1983; Neff, 1986), like those used by Yasin et al. (2013b). In the current study, confusion would be expected to be largest for shortest signals in the on-frequency masking condition and would therefore be expected to cause the amount of compression to be *overestimated*. Confusion effects can therefore not explain why, in the current study, the masker level at threshold as a function of signal duration grows differently at medium levels than in Yasin et al. (2013b, 2014). However, the similarity in the estimates of cochlear gain and cochlear-gain reduction suggest that the cause of this difference does not detract from the main findings of this study.

As in Chapter 3, no correlation was found between FDMC estimates of maximum cochlear gain and absolute hearing thresholds or between FDMC estimates of cochlear-gain reduction by the elicitor and increase

in absolute threshold caused by the elicitor. The only correlation that reached significance was between cochlear-gain reduction estimated psychophysically and OAE IO suppression for the contra-long elicitor. However, in the current study, a reduction in cochlear gain was only estimated for two of the five participants, so this correlation was based on very little data. More participants would be required for it to be concluded with confidence that a correlation exists in this condition.

As in Chapter 3, estimates of maximum cochlear gain varied substantially across participants (by ~21 dB), which supports the suggestion that the amount of cochlear gain may vary substantially, even in normal hearing listeners. Further research is required to establish whether this widely differing cochlear gain across normal-hearing listeners is predictive of susceptibility to hearing loss caused by noise trauma or ageing and whether it may be a factor in cases of obscure auditory dysfunction (see Chapter 3, Section 3.4). Furthermore, the wide variation in the effect of the ipsilateral elicitors across participants (from 6.4 dB to 19.5 dB for the ipsi-long elicitor and from 9.7 dB to 20.5 dB for the short elicitor) suggests that whichever mechanism was causing these effects (temporal suppression or MOCR) also varies greatly across normal-hearing listeners.

4.5 CONCLUSION

Yasin et al. (2014) measured a large psychophysical effect on FDMCs caused by a long-duration noise elicitor, which they attributed to MOCR-induced cochlear-gain reduction. This effect was replicated in the current study. An elicitor that was thought to be too short to elicit the MOCR, but that produced the same amount of masking as the long elicitor used by Yasin et al. (2014), was found to evoke the same psychophysical effect, indicating, either, that the time course of the MOCR is faster than previously thought, or, that the effect was not due to MOCR-induced cochlear-gain reduction. It is argued that the effect of the ipsi-long and ipsi-short elicitors may both, at least partially, be caused by temporal suppression. The findings of the current study suggest that the approach used by Yasin et al. (2014) cannot give a quantitative estimate of MOCR effects under conditions where the elicitor is presented shortly before the masker. A further condition was measured whereby the long elicitor, which had been presented to the ear in which the cochlear-gain reduction was measured, was presented to the opposite ear. If the effect of the elicitor used by Yasin et al. (2014) had been due to MOCR-induced cochlear-gain reduction, then this elicitor would also have been expected to cause a measurable reduction in cochlear gain. However, despite producing a significant OAE suppression effect, no effect of the contralateral elicitor was measured in the psychophysical experiment. Because of the narrower bandwidth and lower amplitude of the elicitor used in the current study, the absence of a measurable psychophysical

effect by the contralateral elicitor is consistent with the findings of Chapter 3.

CHAPTER 5

GENERAL DISCUSSION

The aim of this thesis was to establish whether any of the putative perceptual correlates of MOCR-induced cochlear-gain reduction can provide a quantitative measure of the MOCR. The experiment presented in Chapter 2 investigated the overshoot effect, in which a brief signal presented at the onset of a masker is harder to detect when the masker is preceded by silence than when it is preceded by a “precursor” sound (which is often the same as the masker). It has been proposed that, in off-frequency overshoot, the precursor reduces cochlear gain by eliciting the MOCR and thereby causes a reduction in suppressive masking of the signal. In the experiment presented in Chapter 2, overshoot was measured for a masker and precursor that were above the signal in

frequency (off-frequency overshoot). A forward-masking paradigm was used to measure the amount of suppressive masking that the masker subjected the signal to, with and without the precursor present. While the precursor yielded strong overshoot, and the masker produced strong suppression, the precursor did not appear to cause any reduction (adaptation) of suppression. Predictions based on an established model of the cochlear IO function indicate that the failure to measure any reduction in suppression is unlikely to have represented a false negative outcome. These findings suggest that off-frequency overshoot is not a perceptual correlate of MOCR-induced cochlear-gain reduction, and so the overshoot effect is unsuitable as the basis for a quantitative measure of the MOCR.

Enhancement describes the phenomenon whereby a spectral region in a complex sound becomes more salient when that region is preceded by its spectral complement (a precursor). Because of the similarity between overshoot and enhancement, they have been attributed to similar mechanisms (Viemeister and Bacon, 1982; Strickland, 2008). However, in enhancement there is clear evidence that the precursor causes an increase in the signal response (Viemeister and Bacon, 1982; Byrne et al., 2011), whereas the results presented from Chapter 2 indicate that no such increase occurs in off-frequency overshoot. This suggests that off-frequency overshoot is based on a different mechanism to enhancement.

As discussed in Chapter 2, it is possible that overshoot, rather than being caused by an MOCR-induced cochlear-gain reduction, is due to more central processes related to selective attention. Carlyon (1989) argued that overshoot may be the result of the precursor releasing the masker and signal from perceptual grouping (Koffka, 1922; Dannenbring and Bregman, 1978; Darwin, 1984) by effectively extending the masker and shifting its onset away from the signal onset. Viemeister et al. (2013) tested whether grouping can explain enhancement by comparing the amount of enhancement for precursors that had either the same, or a different, perceptual quality to the masker. If Carlyon's grouping hypothesis does apply to enhancement, a precursor that sounds different from the masker, and would therefore be less effective at ungrouping the masker and signal, should yield little, or at least less, enhancement. Contrary to this prediction, Viemeister et al. (2013) found that the amount of enhancement was similar irrespective of whether the precursor and masker sounded the same or different. It was therefore concluded that enhancement is not caused by perceptual grouping. It remains possible, however, that perceptual grouping plays a role in overshoot. This could be tested in a future study by, as in Viemeister et al. (2013), comparing the size of the overshoot effect for precursors that either have the same, or a different, perceptual quality to the masker.

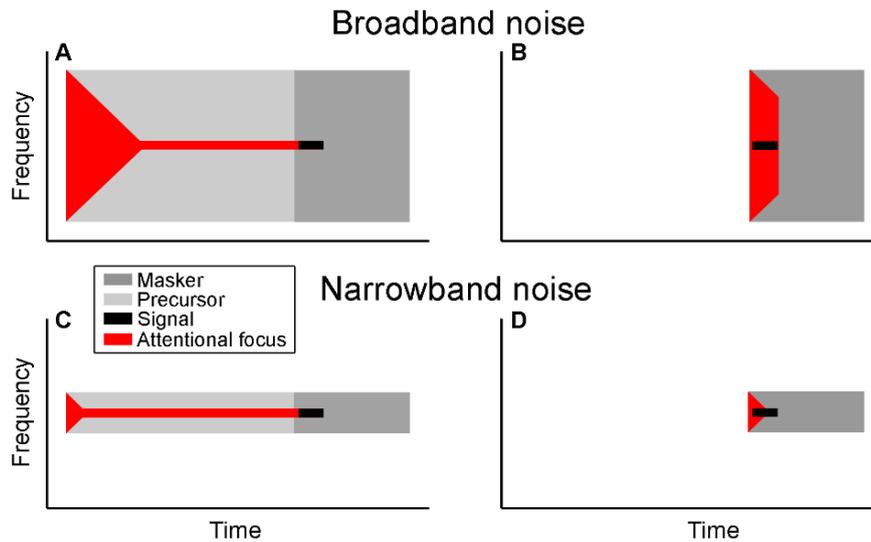


Figure 5.1: A schematic representation of the diversion of attention hypothesis applied to conditions of frequency certainty (see text). Overshoot with both a broadband-noise masker and precursor (panels A and B) and a narrowband-noise masker and precursor (panels C and D) is shown. Conditions with (panels A and C) and without (panels B and D) the precursor are shown. The shaded red area represents the frequency range in which attention is focused up until the signal offset.

Another central mechanism, suggested in Chapter 2 (Section 2.3), which may explain overshoot, is “diversion of attention” (Scharf et al., 2008). Under this hypothesis, the masker draws attention away from the signal frequency towards the masker frequency; continuous presentation of the precursor and masker removes the masker onset and thus eliminates this effect, making the signal easily detectable. This idea is shown schematically in Figure 5.1 (upper panels). When the precursor is not present (panel B), the onset of the broadband-noise masker diverts

attention across a wide range of frequencies, making the signal harder to detect; when the precursor is present, however, (panel A) the listener has time to refocus attention at the signal frequency. A similar diversion of attention away from the signal frequency could be caused by the off-frequency masker used in Chapter 2. In order to test the diversion of attention hypothesis, Scharf et al. (2008) measured overshoot with a broad- or narrow-band-noise masker and precursor (centred at the signal frequency) under conditions where participants either did or did not know which frequency the signal would be presented at (frequency certainty or uncertainty, respectively). They found that frequency uncertainty reduced the amount of overshoot for a broadband-noise masker and precursor, but that, for a narrowband-noise masker and precursor, overshoot was only seen under conditions of frequency uncertainty. For overshoot with a broadband-noise masker and precursor, Scharf et al. (2008) argued that the beneficial effect of the precursor was absent under the conditions of frequency uncertainty, because listeners were unable to focus attention on the unknown signal frequency. For overshoot with a narrowband-noise masker and precursor, they suggested that, under conditions of frequency certainty, the masker did not draw attention away from the signal frequency because it only contained frequency components near the signal frequency (Figure 5.1, lower panels). The precursor was therefore not beneficial for signal detection and created no overshoot. However, when the signal frequency was uncertain, it was argued, the precursor acted as

an attentional cue to the signal frequency and therefore increased signal detectability. A future study could examine whether frequency certainty and uncertainty have a similar effect on tonal maskers and precursors that are either at the signal frequency, or above the signal frequency, as in off-frequency overshoot. A masker and precursor centred at the signal frequency would be expected to act like the narrowband noises in Scharf et al.'s experiment and a masker and precursor above the signal frequency like the broadband noises, with the masker diverting attention away from the signal frequency (in the latter condition, the frequency separation between the precursor and the signal would need to be randomised, so that the precursor could not cue the signal frequency remotely). The results from the proposed experiment would help to establish whether overshoot for tonal maskers and precursors may also be the result of diversion of attention.

In Chapter 3, an approach for obtaining a quantitative estimate of MOCR-induced cochlear-gain reduction by a contralateral noise in humans was refined. Cochlear gain and cochlear-gain reduction were measured using a modified version of the temporal masking curve (TMC) method proposed by Nelson et al. (2001). Using this approach, a contralateral broadband-noise elicitor was found to produce a highly significant effect on both on- and off-frequency masker levels at threshold. This effect was well-predicted by fitting a generic model of the cochlear IO function and assuming that the elicitor effect was to reduce cochlear gain. It was

shown that the precursor effect could be measured using only the on-frequency TMC.

Unlike OAE suppression measurements, the approach developed in Chapter 3 produces a quantitative estimate of MOCR-induced reduction in cochlear gain (see Section 3.4). It could therefore have important translational and basic-science applications. In a basic-science setting, the approach could be used to make parametric measurements of the MOCR in humans. For example, it could be used to measure the effect of the elicitor bandwidth, and the results could be compared with corresponding OAE suppression measurements. Such a study, as well as shedding light on the functional importance of the MOCR, could be critical for understanding the limitations of OAE suppression measurements. In a clinical setting, the approach might be useful as an audiometric profiling tool, particularly in older listeners in whom OAEs, which are frequently used for audiometric profiling, are often small or not measurable (Keppler et al., 2010).

As suggested in Chapter 3, individual differences in the amount of gain applied by the cochlear amplifier in audiometrically normal listeners might give insights into the underlying cause of obscure auditory dysfunction. Obscure auditory dysfunction refers to the fact that some listeners with audiometrically normal hearing report difficulties understanding speech in noisy environments. A future study could explore whether estimates of cochlear gain and MOCR-induced

reduction in cochlear gain made using the approach developed in Chapter 3 correlate with estimates of obscure auditory dysfunction. Individual differences in cochlear gain or MOCR function may also contribute to some cases of auditory processing disorder. Auditory processing disorder (which may be related to obscure auditory dysfunction) is typically used as an umbrella term, referring to cases of listening difficulties, often resulting in impaired language learning, that have no known peripheral origin (Cacace and McFarland, 1998, 2005; Fey et al., 2011). As for obscure auditory dysfunction, people with auditory processing disorder may exhibit deficits in cochlear gain or MOCR function that may not be apparent in standard audiometric measurements (see Section 3.4).

Individual differences in MOCR function might account for differences in susceptibility to noise trauma or age-related hearing problems and the approach for measuring MOCR function developed in Chapter 3 might be useful for predicting the likelihood of a given individual incurring these problems. This idea is supported by findings from Maison and Liberman (2000), discussed in Chapter 1 (Section 1.2.1), which have shown evidence that, in animals, MOCR strength is predictive of susceptibility to damage caused by noise exposure. As suggested in Chapter 3, a predictive measure of susceptibility to noise trauma might help to preserve healthy hearing by influencing lifestyle choices and encouraging preventative behaviours.

An interesting and potentially important finding of Chapter 3 is that, unlike the results from the psychophysical measurements, the results from the OAE suppression measurements were sensitive to the order in which the with- and without-elicitor conditions were measured. OAE amplitude suppression by the elicitor was only found when the with- and without-elicitor conditions were interleaved within the same session. The order effect found in the experiment presented in Chapter 3 might be related to that found by Micheyl and Collet (1996). They measured a significant correlation between OAE amplitude suppression and improvement in signal-in-noise audibility by a contralateral elicitor only when the with-elicitor condition preceded the without-elicitor condition. The fact that, in the experiment presented in Chapter 3, the order effect was present in the OAE, but not the psychophysical, data suggests that the effect is specific to OAEs and thus not related to MOCR-induced reduction in cochlear gain. The effect may thus represent an important confound when measuring MOCR-induced gain reduction using only OAEs. Further study is required so that the underlying cause(s) of this order effect can be understood.

Chapter 4 expanded on recent findings by Yasin et al. (2014). They measured a large psychophysical effect caused by a long-duration noise elicitor presented ipsilateral to the signal and the masker, which they attributed to MOCR-induced cochlear-gain reduction. The aim of the experiments presented in Chapter 4 was to establish whether the large effect measured by Yasin et al. (2014) truly represents MOCR-induced

reduction in cochlear gain. Yasin et al. (2014) used a modified version of the TMC method, known as the fixed-duration masking curve (FDMC) method and controlled for any direct masking of the signal by the elicitor by adjusting the signal level. In one of the experiments presented in Chapter 4, the effect of a long-duration ipsilateral elicitor, like the one used by Yasin et al. (2014), was replicated and compared to the effect of an elicitor that produced the same amount of masking of the signal, but was thought to be too short to elicit the MOCR in time to affect the masker. The short elicitor evoked a similar or larger psychophysical effect than the long elicitor, indicating, either, that the MOCR acts more quickly than previously thought, or, that the effect was not due to MOCR-induced cochlear-gain reduction. In Chapter 4, it is argued that the effects of the long and short elicitors may have both been caused by direct nonlinear interactions due to partial temporal overlap between the cochlear responses to the elicitor and masker. The results of Chapter 4 suggest that direct cochlear interactions represent a major confound when using ipsilateral elicitors presented shortly before the masker.

In another experiment presented in Chapter 4, the effect of the long elicitor was also measured when it was presented contralateral to the signal and masker. If the large effect of the ipsilateral elicitor had been due to MOCR-induced cochlear-gain reduction, then the contralateral elicitor would also have been expected to induce a measurable reduction in cochlear gain. Contrary to this expectation, no effect of the contralateral elicitor on FDMCs was found. This makes sense when

considered in relation to the findings of Chapter 3, where the elicitor was also presented contralaterally but had a much broader bandwidth and was presented at a higher level. The elicitor used in Chapter 3 produced an effect of ~ 4 dB on average. OAE suppression estimates by Lilaonitkul and Guinan (2009a) suggest that the narrower bandwidth would approximately halve the amount of MOCR-induced gain reduction. This halving of the expected effect size due to elicitor bandwidth, coupled with a reduction in the effect size due to the lower elicitor level used in Chapter 4, could account for the absence of a measurable effect in the FDMC measurements. The contralateral elicitor did produce a significant effect in the OAE suppression measurements. However, this was probably due to the fact that, in the OAE measurements, the elicitor had a broader bandwidth than in the FDMC measurements and would therefore have been expected to produce a larger cochlear-gain reduction. A broader-band elicitor was used because the probe click was more broadband than the tonal signal in the FDMC experiments and the OAE spectrum was expected to contain little energy at the signal frequency.

In Chapter 4, the data were analysed using a similar cochlear IO function model as was used in Chapter 3. The fit produced in Chapter 4 was not as good as that in Chapter 3. This appears to be due to two factors. Firstly, there was a great deal of variability in the off-frequency masker level at threshold both within and across participants, and secondly, the on-frequency FDMC initially grew more slowly than the off-frequency FDMC,

which was unexpected. The implication of the unexpectedly slow growth in the on-frequency masker level at threshold is that, for low input levels, the cochlear IO function is expansive, that is, the output grows faster than the input. As discussed in Chapter 4 (Section 4.4), this has also been found in other studies using the FDMC method (Yasin et al., 2013b, 2014), but is not consistent with psychophysical studies using other methods (e.g., Oxenham and Plack, 1997; Nelson et al., 2001) or with physiological data from animals (Robles and Ruggero, 2001). The compression estimates, without the elicitor present, made using the FDMC method are also not consistent with these other studies. This suggests that the crucial assumption in the FDMC method, that on- and off-frequency masking decays at the same rate at the signal frequency as a function of signal duration, may not hold. A further study is needed to test this assumption.

Although there are difficulties associated with quantitatively measuring MOCR-induced cochlear-gain reduction by an ipsilateral elicitor in humans, it is important that such a measure is identified. If it is found that MOCR-induced cochlear-gain reduction by an elicitor presented contralateral to a probe is much weaker than that by an ipsilateral elicitor, as the results of Lilonaitkul and Guinan (2009a) suggest may be the case for sounds with a narrow bandwidth, then this could have significant implications for processing of spatial localisation cues. It has been argued that the absence of a strong link between the amounts of gain applied to a sound in each ear might lead to distortion of ILD cues

(Byrne and Noble, 1998). However, others have argued that, particularly in complex acoustic environments, the quality of ILD cues is not improved by linking the amounts of gain applied (Byrne and Noble, 1998; Moore, 2008; Kreisman et al., 2010; Mullin, 2010; Arweiler, 2011; Wiggins and Seeber, 2013). Because cochlear gain affects the response latency of the cochlea (discussed in Section 1.2.2; see Francis and Guinan, 2010), the absence of a strong contralateral MOCR might also be expected to distort ITD cues. As discussed in Section 1.2.2, it has been argued that any detrimental MOCR effects on localisation cues might be compensated for at later stages of auditory processing. However, the role of the MOCR in spatial hearing remains largely unknown and it remains possible that the MOCR may somehow facilitate spatial hearing (Francis and Guinan, 2010).

In both Chapter 3 and Chapter 4, there is evidence of a correlation between the psychophysical estimates of MOCR-induced cochlear-gain reduction by a contralateral elicitor and OAE IO suppression estimates, but no evidence of a correlation was found for ipsilateral elicitors. The ipsilateral elicitor effects may be confounded by temporal suppression effects, which would have affected the OAE and psychophysical estimates differently, as discussed in Chapter 4 (Section 4.4). No evidence of a correlation between psychophysical estimates and OAE amplitude suppression estimates was found. This may be because, for a contralateral elicitor, OAE IO suppression is more robust than OAE amplitude suppression due to factors unrelated to cochlear gain, such as

differences between the anatomical properties of the ear-canal or middle ear (Probst et al., 1991) or effects of gender (Moulin et al., 1993b). The correlations may have been stronger if the OAE and psychophysical measurements had been made under more similar conditions. OAEs were evoked by a click and therefore, unlike the psychophysical estimates, measured the combination of MOCR effects at multiple frequencies (although the largest MOCR effects on OAEs are likely to have occurred near the peak of the OAE spectrum, which, in Chapter 3, was near the signal frequency used in the psychophysical measurements). Furthermore, OAEs were measured under passive listening conditions, with participants watching a silent movie, whereas the psychophysical measurements required active listening. Thus, if the MOCR is under attentional control, as some studies suggest (see Section 1.2.2), then this may also make a correlation between OAE and psychophysical estimates difficult to measure. An important challenge for future research in this area is to more closely match psychophysical and OAE measurements, possibly by integrating them (i.e., measuring OAEs whilst the participant is performing a psychophysical task).

One puzzling finding in Chapter 4 was that the short ipsilateral elicitor produced a large OAE IO suppression, but the long ipsilateral elicitor did not produce any IO suppression. This could indicate that the effects of the short and long elicitors have different underlying mechanisms, although further research is required for this to be established. As discussed in Section 4.4, the fact that an effect of the short elicitor was

also seen for a short elicitor in the psychophysical measurements of Chapter 4 may also indicate that the response time of the MOCR is faster than previous thought. Another puzzling finding is that the contralateral elicitor used in Chapter 3 did not, on average, produce any IO suppression, yet the elicitor used in Chapter 4 did. It is not clear why the differences in the way the OAEs were measured in these experiments, such as the elicitor being presented continuously in Chapter 3 and for only 500 ms in Chapter 4, would lead to reduced IO suppression in Chapter 3. One possibility is that when the continuous noise elicitor was presented there was an adaptation (reduction) of the elicitor effect over time. However, van Zyl et al. (2009) found no evidence of such an adaptation effect over the course of a fifteen-minute long elicitor.

It is possible that attentional factors added variability to the psychophysical estimates of cochlear gain and cochlear-gain reduction. For example, following the idea of diversion of attention, discussed above in relation to the overshoot effect, it is possible that, in the FDMC and TMC measurements (Chapters 3 and 4), the off-frequency masker diverted attention away from the signal frequency and thereby made the signal harder to detect. The on-frequency masker, on the other hand, might attract attention towards the signal frequency, making it easier to detect. Such an attentional effect would reduce the difference between the on- and off-frequency masker levels at threshold and thus lead to the amount of cochlear gain being underestimated. This could be tested by measuring FDMCs and TMCs under conditions of frequency uncertainty

as in Scharf et al. (2008). However, it seems unlikely that attentional factors have a large effect on cochlear gain estimates for either the FDMC or TMC methods, as both have produced estimates that accord well with physiological findings from animals (see Chapters 3 and 4; Robles and Ruggero, 2001; Yasin et al., 2013b).

A significant limiting factor in the experiments associated with Chapters 3 and 4 is the MEMR. When the MEMR is activated, it can cause a reduction in the cochlear response that could be mistaken for a reduction in cochlear gain. The MEMR would be expected mainly to affect OAE estimates, because it has little effect above ~ 2 kHz (Nuttall, 1974; Rosowski and Relkin, 2001), although it may also affect the off-frequency masker levels at threshold in Chapter 3. To ensure that the elicitors used in Chapters 3 and 4 would not elicit the MEMR, they were presented at a level below the MEMR threshold. This meant that they probably did not maximally elicit the MOCR. In future studies, it may be possible to control for elicitation of the MEMR by performing concurrent electromyographic (EMG) measures to monitor MEMR activation. This would allow the elicitor to be set at the maximum level possible in each participant, without the MEMR being elicited. Alternatively, it is possible that stimulus-frequency OAEs, in which pure-tone probe sounds are used to measure more frequency-specific effects, may permit the use of higher elicitor levels (Guinan et al., 2003), although these effects are often very small and therefore require a large number of measurements. It is also possible that other measurement techniques may give independent

verification that psychophysical effects are due to the MOCR, whilst permitting higher elicitor levels to be used. For example, it may be possible to develop a method for measuring MOCR-induced cochlear-gain reduction using auditory brainstem responses, based on the approach for measuring cochlear gain and compression presented by Krishnan and Plack (2009). They compared the latency of the wave-V response to a short, 4 kHz tonal signal with a forward-masker either at or below the signal frequency. Wave V of the auditory brainstem response represents neural activity from the lateral lemniscus and/or inferior colliculus that is synchronised to the onset of a brief stimulus (Moller and Jannetta, 1982). By measuring the wave-V latency as a function of masker level, and assuming that wave-V latency is an index of the masker's forward-masking effectiveness (see Kramer and Teas, 1982; Lasky and Rupert, 1982; Ananthanarayan and Gerken, 1983, 1987), Krishnan and Plack (2009) were able to construct a cochlear IO function using similar assumptions to those in the TMC method used in Chapters 3. They estimated a maximum cochlear gain of 15.5 dB and a compression exponent of 0.25. These estimates are broadly in line with the psychophysical estimates presented in Chapter 3 and those from previous human and animal studies, when it is considered that the lowest masker level they were able to use was 50 dB SPL.

This thesis has made contributions towards the understanding of two auditory phenomena and has improved existing psychophysical methods for measuring cochlear gain, cochlear compression, and MOCR-induced

cochlear-gain reduction in humans. In this thesis, evidence has been presented against the role of the MOCR in overshoot. Evidence has also been put forward suggesting that there is a fundamental difference between overshoot and enhancement, which had previously been thought to be caused by similar mechanisms. The findings of this thesis have also cast doubt over whether a recent finding of a large psychophysical effect on cochlear-gain estimates is due to the MOCR. Progress has also been made towards the core aim of the thesis, which was to identify a quantitative measurement of the MOCR. An existing psychophysical method for estimating cochlear gain, cochlear compression and MOCR-induced cochlear-gain reduction was modified and a mathematical model was developed which allowed these estimates to be made more reliably and efficiently. There is an exciting possibility that this approach to measuring cochlear gain and MOCR-induced cochlear-gain reduction could become a valuable audiometric profiling tool and give insights into the individual differences that underlie hearing problems in audiometrically normal listeners. It is also possible that parametric exploration of the MOCR using this approach will, in the near future, allow the functional importance of the MOCR in humans to be properly understood.

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APPENDIX A

Table 1: Model estimates of G_{max} , c , BP_1 , and P , all without the elicitor present and ΔG are shown for each individual and averaged across all 12 participants.

	G_{max} (dB)	c	BP_1 (dB)	P (dB)	ΔG (dB)
L1	16.5	0.43	29.2	25.7	2.3
L2	26.1	0.15	28.5	29.6	0
L3	36.0	0.12	23.8	13.9	0
L4	29.4	0.18	32.1	14.7	3.6
L5	10.7	0.23	33.4	34.2	2.3
L6	36.1	0.19	30.0	12.3	4.6
L7	11.4	0.56	27.8	33.2	11.4
L8	29.2	0.14	29.6	11.1	0
L9	30.5	0.16	35.7	8.7	0.6
L10	31.2	0.08	28.3	9.3	0.9
L11	23.3	0.38	30.5	22.7	4.1
L12	18.1	0.65	19.6	23.7	18.1
Mean	24.9	0.27	29.0	19.9	4.0
($\pm SE$)	(± 2.56)	(± 0.054)	(± 1.22)	(± 2.71)	(1.58)

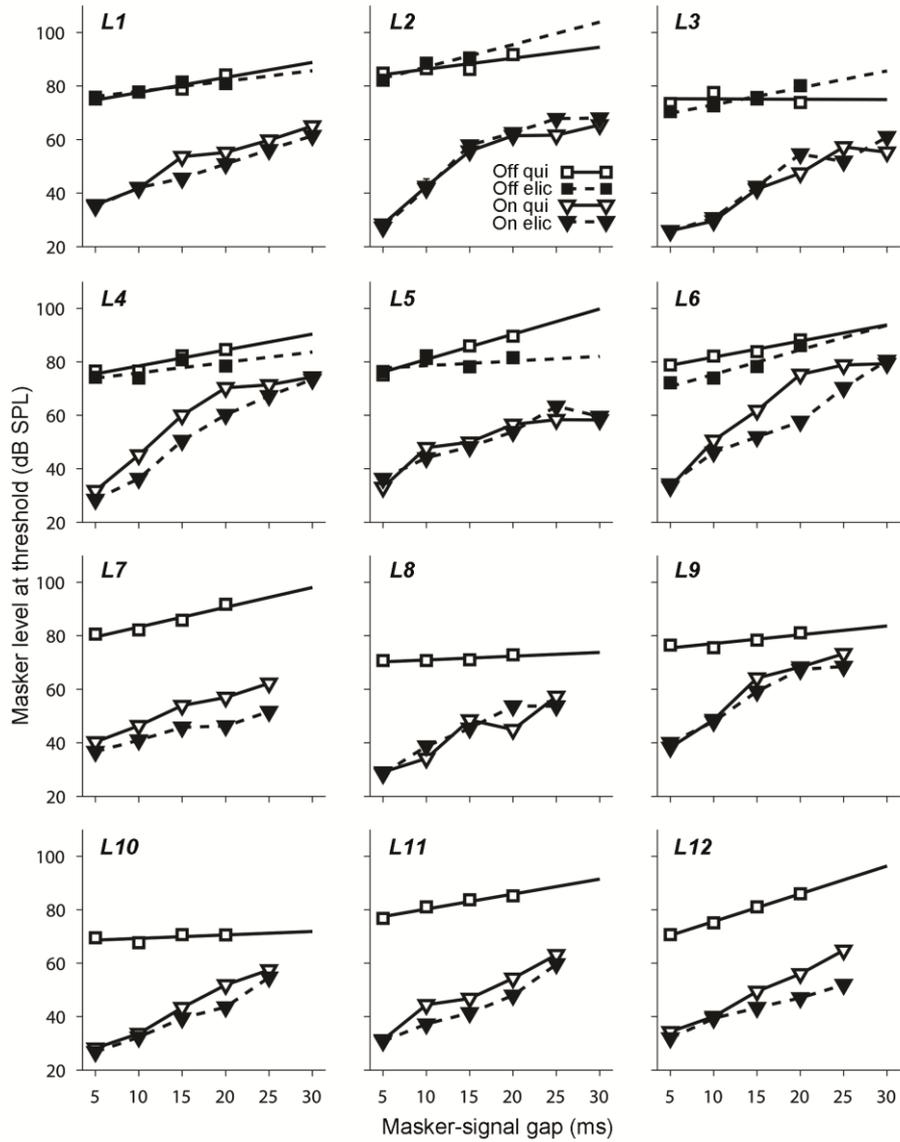


Figure 1: On- and off-frequency TMCs with (filled upside-down triangles and squares, respectively) and without (open upside-down triangles and squares, respectively) the elicitor present, for each of the 12 participants. The error bars show the standard error of the mean.

APPENDIX B

Table 1: Model estimates of G_{max} , c , and BP_1 , all without the elicitor present and ΔG for the ipsi-long (ΔGIL), ipsi-short (ΔGIS), and contra-long (ΔGCL) elicitors. Values are shown for each individual and averaged across all five participants.

	G_{max} (dB)	c	BP_1 (dB)	ΔGIL (dB)	ΔGIS (dB)	ΔGCL (dB)
<i>L1</i>	26.6	0.34	50.5	19.5	20.5	4.3
<i>L2</i>	9.7	0.27	39.7	9.7	9.7	3.5
<i>L3</i>	20.4	0.23	46.5	9.9	18.3	0
<i>L4</i>	30.8	0.28	33.8	7.9	12.2	0
<i>L5</i>	23.2	0.73	48.4	6.4	9.8	0
Mean	22.1	0.37	43.8	10.7	14.1	1.6
($\pm SE$)	(± 3.57)	(± 0.091)	(± 3.07)	(± 3.94)	(± 3.50)	(± 4.02)

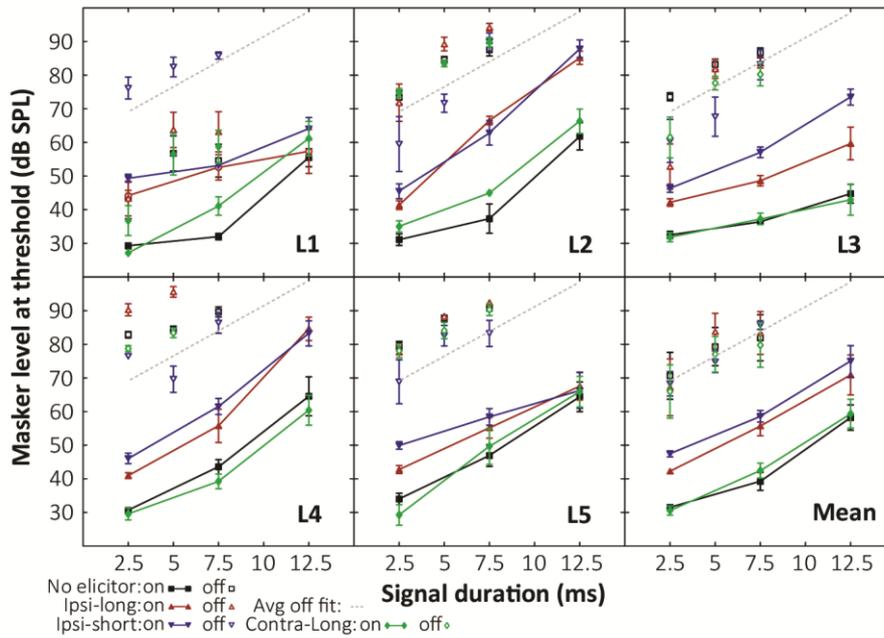


Figure 1: Individual and average masker levels at threshold for the on-frequency masker (filled symbols and solid lines) and the off-frequency masker (open symbols and no lines) are shown for each participant as a function of the signal duration. The dashed lines show the linear regression fit of the average off-frequency masker levels at threshold. Thresholds without the elicitor, and with the ipsi-long, ipsi-short and contra-long elicitors are shown in black, red, blue, and green, respectively. Error bars show the standard error of the mean.